NTAL
RARY

JUL 17 1946

Volume 25 Number 3

JOURNAL
of
DENTAL RESEARCH

FOUNDED BY WILLIAM J. GIES IN 1919

Official Publication of the

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

June, 1946

The Editor's Viewpoint 105

Dental Status of 71015 Naval Personnel at First Examination in 1942. *C. A. Schlack, J. S. Restarski* and *E. F. Dochterman* (National Institute of Health, Bethesda, Maryland) 107

Clinical and Bacteriological Studies of the Use of a Fluoride Dentifrice. *Edward O. Shaner* (Washington, D. C.) and *R. Reed Smith* (Springfield, Mo.) 121

Decay of the Deciduous Teeth of Formosa Chinese. An Analytical Summary and Interpretation of Certain Statistics Published by Maruyama. *V. O. Hurme* (University of Illinois) 127

Hydrolyzed Saliva As a Source of Nutrients Essential for the Production of Acid by an Oral Strain of Lactobacillus Acidophilus. *David Weisberger* (Harvard University) 137

The Appositional Life Span of the Enamel and Dentin-Forming Cells. I. Human Deciduous Teeth and First Permanent Molars. *Maury Massler* and *Isaac Schour* (University of Illinois) 145

International Association for Dental Research. Proceedings of the Twenty-Fourth General Meeting, Chicago, Ill., March 16 and 17, 1946. Compiled by *Hamilton B. G. Robinson*, Editor (Ohio State University) 151

I. Inaugural address. *Samuel W. Chase*, Ph.D. (Western Reserve University) 151

II. Index of participants and sequence numerals for corresponding abstracts 155

III. First Session: Morning, March 16; abstracts 1–6 155

IV. Second Session: Afternoon, March 16 (joint program with materials section); abstracts 7–10 158

V. Third Session: Evening, March 16

A. Materials section; abstracts 11–16 159

B. General session; abstracts 17–25 161

VI. Fourth Session: Morning, March 17; abstracts 26–36 165

VII. Fifth Session: Afternoon, March 17; abstracts 37–45 169

VIII. Sixth Session: Evening, March 17; Annual Dinner; abstracts 46–49 173

IX. Abstracts of papers read by title; 50–86 173

X. Executive proceedings and register of newly elected officers and members 187

PUBLISHED BI-MONTHLY AT MT. ROYAL AND GUILFORD AVENUES, BALTIMORE 2, MD.

By THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

EDITORIAL OFFICE: Ohio State University, College of Dentistry, Columbus 10, Ohio.

Copyright 1946, by the International Association for Dental Research

Made in United States of America

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

EDITORS OF THE JOURNAL OF DENTAL RESEARCH

Editor Emeritus—William J. Gies

Publication Committee: J. Frank Hall, S. S. Arnim and Hamilton B. G. Robinson.

Editor—Hamilton B. G. Robinson. *Business Manager*—J. Frank Hall. *Circulation Manager*—S. S. Arnim.

Assistant to the editor—Paul C. Kitchin and J. B. Brown. *Assistant to the business manager*—Henry M. Swenson. *Assistant to the circulation manager*—J. William Adams.

Board of Editors—Editor-emeritus, members of the Publication Committee and sectional representatives named in fifth column below.

THE SECTIONS—THEIR SECRETARIES, AND REPRESENTATIVES IN THE COUNCIL AND IN THE BOARD OF EDITORS (1941)

Division	*Section*	*Secretary*	*Councillor*	*Editor*
Canada	Halifax	S. G. Ritchie	J. S. Bagnall	S. G. Ritchie
	Toronto	A. W. Ellis	A. D. Mason	Thos. Cowling
	Winnipeg	E. Roy Bier	H. J. Merkeley	E. Roy Bier
China	Chengtu	R. Gordon Agnew	R. Gordon Agnew	R. Gordon Agnew
England	London	Evelyn Sprawson	Evelyn Sprawson	Evelyn Sprawson
Czechoslovakia	Prague	Friedr. Neumann	Friedr. Neumann	Jan Jesensky
Germany	Vienna	Fritz Driak	R. Trauner	F. Shönbauer
Hungary	Budapest	Ferenc Köszeg	———	———
Palestine	Palestine	S. Lewin-Epstein	S. Lewin-Epstein	S. Lewin-Epstein
South Africa	Johannesburg	Julius Staz	J. N. Noriskin	W. B. Osborn
United States	Ann Arbor	Philip Jay	Philip Jay	Philip Jay
	Baltimore	M. S. Aisenberg	E. C. Dobbs	J. B. Robinson
	Boston	G. E. Thompson	B. G. Bibby	———
	Chicago	R. M. Stephan	A. G. Brodie	———
	Cleveland	S. W. Chase	Thomas J. Hill	S. W. Chase
	Columbus	L. F. Edwards	J. H. Kaiser	H. D. Spangenberg, Jr.
	Detroit	Samuel J. Lewis	P. C. Lowery	Samuel J. Lewis
	Houston	F. C. Elliot	F. C. Elliot	F. C. Elliot
	Indianapolis	Henry M. Swenson	Maynard K. Hine	R. W. Phillips
	Iowa City	A. O. Klaffenbach	A. W. Bryan	George S. Easton
	Louisville	Raymond E. Myers	———	H. T. Knighton
	Minnesota	G. A. Montelius	P. J. Brekhus	K. W. Ray
	New Orleans	J. T. Ginn	S. L. Tiblier	F. J. Wolfe
	New York	Isaac Neuwirth	L. M. Waugh	S. C. Miller
	Philadelphia	Thomas J. Cook	J. L. T. Appleton	J. L. T. Appleton
	Pittsburgh	L. E. Van Kirk	H. E. Friesell	W. H. Wright
	Richmond	A. P. Little	S. S. Arnim	Harry Lyons
	Rochester	H. B. McCauley	H. B. McCauley	P. P. Dale
	San Francisco	S. Epstein	W. C. Fleming	Herman Becks
	St. Louis	W. B. Gurley	William Bauer	L. R. Boling
	Tennessee	———	O. A. Oliver	M. T. Dean
	Washington	H. W. Krogh	F. S. Arnold	B. E. Erickson
Materials Group		F. A. Peyton	E. W. Skinner	N. O. Taylor

OFFICERS OF THE ASSOCIATION

President—Samuel W. Chase, *President-elect*—Harold C. Hodge, *Vice-president*—A. G. Brodie, *Secretary-treasurer*—Edward H. Hatton, *Editor*—Hamilton B. G. Robinson, *Trustees*—R. W. Bunting, Edward H. Hatton, Thomas J. Hill, Leuman M. Waugh, J. L. T. Appleton.

Subscription price $5.00 per volume. Single numbers when available $1.00.

Entered as second-class matter June 24, 1919, at the Post Office at Baltimore, Maryland, under the act of March 8, 1879

WAVERLY PRESS, INC.

BALTIMORE, U. S. A.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Organized: New York City, Dec. 10, 1920. Of those at the meeting of organization, the following remain active members: Adolph Berger, Theodor Blum, G. W. Clapp, H. S. Dunning, W. J. Gies, R. G. Hutchinson, Jr.; T. P. Hyatt, Milo Hellman, A. H. Merritt, B. B. Palmer, P. R. Stillman, L. M. Waugh.

General Meetings have been held on this schedule: (1) New York, Dec. 21, '22. (2) Chicago, Mar. 7, '24. (3) Chicago, Mar. 20, '25. (4) Chicago, Mar. 26, '26. (5) New York, Apr. 19, '27. (6) Washington, Mar. 25–28, '28. (7) Chicago, Mar. 23–24, '29. (8) Toronto, Mar. 24–26, '30. (9) Memphis, Mar. 21–22, '31. (10) Columbus, Mar. 19–20, '32. (11) Chicago, Mar. 18–19, '33; adjourned executive session, New York, May 25, '33. (12) Chicago, Mar. 17–18, '34. (13) Chicago, Mar. 16–17, '35. (14) Louisville, Mar. 14–15, '36. (15) Baltimore, Mar. 13–14, '37. (16) Minneapolis, Mar. 12–13, '38. (17) Cleveland, Mar. 18–19, '39. (18) Philadelphia, Mar. 13–14, '40. (19) St. Louis, Mar. 15–16, '41. (20) New York, Mar. 14–15, '42. (21) Chicago, Mar. 13–15, '43. (22) Chicago, Mar. 18–19, '44. (23) Chicago, May 27, '45. (24) Kansas City, Mar. 16–17, '46.

Objects (*Const., Art. II*): . . . "(a) To promote broadly the advancement of active research in all branches of dental science and in related phases of the sciences that contribute directly to the development of dentistry, and which add to the knowledge of the mouth and teeth and of their relations to the body as a whole; . . . (b) to encourage and facilitate coöperative effort and achievement by, and mutual helpfulness among, investigators in all nations in every division of stomatology; (c) to the end, particularly, that dentistry may render cumulatively more perfect service to humanity." (The Association has been using "dentistry" and "stomatology" as synonyms.)

Membership: total (Dec. 1, 1945), 633. *Eligibility* (*Const., Art. 14, Sec. 1*).—"Any person who has conducted, and published an account of, a meritorious original investigation in dental science, or in any of the sciences contributory to stomatology, shall be eligible to membership in this Association, providing such person conforms to the recognized standards of professional ethics." *Election.*—New members may be elected *only at general meetings of the Association.* Membership in a section is secondary to membership in the Association as a whole. Removal of members from one geographic center to another does not affect their membership in the Association.

Endowment fund of the International Association for Dental Research (as of Dec. 1, 1944): $1,370.98. There have been no expenditures from this fund.

Divisions may be organized by the members in any nation. There are now divisions in Canada, China, England, Germany, Hungary, Palestine, South Africa, and the United States—8. *Sections* may be organized by the members in geographic centers within the divisions. There are now sections (32) in Vienna ('29); Halifax ('28), Toronto ('21), Winnipeg ('30); Chengtu ('34); Prague ('32); London ('31); Budapest ('34); Johannesburg ('34); Palestine ('39); Ann Arbor ('23), Baltimore ('33), Boston ('20), Chicago ('20), Cleveland ('30), Columbus ('32), Detroit ('37), Houston ('39), Indianapolis ('41), Iowa City ('36), Louisville ('32), Minnesota ('28), New Orleans ('37), New York ('20), Philadelphia ('28), Pittsburgh ('28), Richmond ('33), Rochester ('33), St. Louis ('28), San Francisco ('24), Tennessee ('44), Washington, D. C. ('31). The names of the secretaries are given on p. 2 of this cover

Journal of Dental Research: The Association's official publication. See register of officers and editors on page 2 of cover.

BOOKS RECEIVED

American Dental Association, Roster of Members (Chicago, Am. D. Assoc., 1946) pp. 343.

Burket, L. W. Oral Medicine (Philadelphia, J. B. Lippincott Company: 1946) pp. 674.

INFORMATION FOR PROSPECTIVE CONTRIBUTORS, SUBSCRIBERS, OR ADVERTISERS

The JOURNAL OF DENTAL RESEARCH was dedicated by its founder to the dissemination of knowledge pertaining to the teeth and mouth, and to their relations to the body as a whole. It publishes original investigations in these and related fields. Articles are accepted only on condition they are contributed solely to this JOURNAL. Articles appearing in other domestic or foreign periodicals will not be reprinted in the JOURNAL except in extraordinary cases. The JOURNAL will supply a limited number of half-tones or zinc etchings without cost to the author. References to the literature must conform to the following style:

1. (To a periodical)—Doe, J. A., Dental disease, *J. D. Res.*, **16**: 42, 1937.
2. (To a book)—Doe, J. A., *The Dental Practice* (Chicago, Smith: 1930), p. 114.

See McBride, *J. D. Res.*, **16**: 537, 1937.

Titles should be given for all references. Papers presented at society meetings should be so footnoted. Reprints, up to 500 copies, will be supplied at prices quoted on request and must be ordered from the printer at the time author's proof is returned. Additional reprints may be purchased only at the discretion of the Board of Editors.

Subscription price for the volume (issued bimonthly beginning with February) is $5.00. Single copies $1.00. The price of volumes and single numbers prior to 1940 will be quoted upon request to the office of the Business Manager.

Address all communications relating to editorial matters to the JOURNAL OF DENTAL RESEARCH, Ohio State University, College of Dentistry, Columbus, 10, Ohio; advertising or business matters to the JOURNAL OF DENTAL RESEARCH, 1121 W. Michigan St., Indianapolis, Ind.

THE EDITOR'S VIEWPOINT

By popular request the Editor's page is being moved anteriorly. Some of our readers complain that it is too hard to find elsewhere.

Drs. Schlack, Restarski and Dochterman present an age and regional distribution study of 71015 sailors (p. 107), fulfilling our hope that the rather complete and very extensive studies of the armed services would soon be released for general use. The greater numbers of dental defects among the New England and Middle Atlantic groups follows the impression of many clinicians but perhaps a more significant finding is the leveling off of regional differences in older age groups. Might this not be considered with Weaver's report (*Brit. D. J.* 88:185, 1944) that differences in caries rates of children raised in fluoride and non-fluoride regions tended to be erased in adults?

Weisberger (p. 137) continues his study of nutrient essentials for *L. acidophilus* in saliva. He appears to have demonstrated that hydrolized saliva supplies certain constituents not available from whole saliva.

Shaner and Smith (p. 121) present evidence that fluorides may effect a reduction in caries incidence in adults. It must be pointed out that group studied was small and some of the bacterial counts were irregular. How many counts are necessary to be considered significant? The report by Permar, Kitchin and Robinson (p. 167) gives the results of an effort to ascertain the answer to this question. We would like to see the results of surface by surface studies of the caries occurrence over a two-year period. Perhaps the authors have projected such a problem.

The contribution from Dr. Hurme (p. 127) is rather different from our usual material. We agree that Maruyama's report on a rather pure ethnic group undisturbed by medical and dental services is worthy of further study.

Drs. Massler and Schour (p. 145) present a theory of the life span of enamel and dentin-forming cells. They use their data to explain dentinogenesis imperfecta, and also imply the relationship between vitamin deficiency and appositional life span.

The Proceeding's of the International Association for Dental Research (p. 151) afford a wealth of material too extensive to discuss. We would like to call your attention to the President's address (p. 151). Too often "addresses" are overlooked as stuffy and trivial. You will find meat in Dr. Chase's remarks. A total of 114 members attended the sessions. In addition there were numerous guests, who are always welcome at the Association's meetings. A total of 48 papers was read at the meetings and an additional 37 were read by title.

A new dental periodical has entered the field under the editorship of Balint Orban. This quarterly publication, *Journal of Endodontia*, will fill a need in a highly specialized field.

Oral Medicine, a textbook by Lester W. Burket is unique. Written by a dentist with a training in medicine acquired under the Rockefeller Fellowship plan at Yale, it is unlike other books in the field. It is not a textbook of diagnosis, for tumors and most surgical diseases are omitted, nor is it a textbook of periodontia because it goes beyond such limits. The various diseases that Burket discusses are not considered in any other single place. The text is thorough and the illustrations invaluable. The "color atlas" is somewhat disappointing because it fails to approach the original pictures in clarity. We believe that the author should avoid use of the term "common" in such sentences as "Leukoplakia . . . is common in pipe smokers" (p. 628). In our experience sodium perborate gives rise to irritative lesions of the oral mucosa *rarely* when used in proper concentration, not "frequently" (p. 624). *Oral Medicine* will be one of the most useful books in our library.

DENTAL STATUS OF 71015 NAVAL PERSONNEL AT FIRST EXAMINATION IN 1942

Age and Regional Distribution of Persons with Specific Type Dental Defects[1]

C. A. SCHLACK, D.D.S., Commander (DC), USN, J. S. RESTARSKI, D.D.S., M.D.S., Commander (DC), USN and E. F. DOCHTERMAN, M.S. Lieutenant (jg), H(W), USNR

From the Naval Medical Research Institute, National Naval Medical Center, Bethesda, Md.

INTRODUCTION

Regional (1, 2) and age (3) differences for dental defects in general have been reported for military personnel. Dental defects have been shown to increase with age in adults of mixed populations by application of indirect methods of analysis (the difference in the dental status of different populations in successive age groups at a given time) (4), and by direct methods (the difference in the dental status of the same populations in successive age groups after a 1-year interval) in military personnel (3). The rate of increase seems to diminish as persons reach the age of forty (3, 4), and, to some extent, varies with different methods of oral examination (3a).

Ferguson (1) summarizing a dental analysis on 4602 naval recruits, reported that men from the New England and Middle Atlantic regions were more dentally defective than were those from the Southern and Western states. Unfortunately arithmetical errors in his table and the smallness of numbers observed from many regions, prevent reliable interpretation. East (5), correlating Army data on "dental rejectees" in the 1863–64 draft with those of the 1918 draft, and these in turn with the naval data of Ferguson, reported a general similarity in trends. However, varying criteria for the dentally defective in drafts of previous wars, make such comparisons of small value. Senn (2) reporting on the dental status of approximately 7000 Army cadets, showed in part, the same geographical trends of dental defects as Ferguson. However, he emphatically does not support Ferguson in the differences between dental defects in persons from the Atlantic and Pacific coasts. Senn found very little difference between these 2 regions, whereas Ferguson supported such differences.

In the studies to which reference has just been made, cumulative scores (sums of the number of decayed, missing and filled teeth, or totals of surfaces involved with caries, fillings, etc.), were used to represent the dental status of each person's

[1] The opinions and views set forth in this article are those of the writers and are not to be considered as reflecting the policies of the Navy Department. Received for publication March 26, 1946.

mouth. In the present study each type dental defect was considered separately. The combination of dental defects under 1 cumulative score was avoided since such scores allow no qualitative analysis. A qualitative and quantitative analysis is of practical importance to the Naval Dental Corps, because it permits more accurate estimates of time factors and personnel required to correct certain of these defects (6).

Previous studies (1, 2, 3, 4, 5) also placed less statistical emphasis upon numbers observed from each region and within each age grouping. In this study much effort was expended on these factors in order to determine more accurately, regional influences on type dental defects.

Fig. 1. States in geographic regions

Dental caries has been assumed the principal cause for teeth to be extracted or for missing teeth in adults by some writers (1, 2, 5). However, evidence presented here may be sufficiently strong to alter such assumptions, particularly when considering young adult age groups and regions in which there appear to be fewer persons with dental defects than in others.

Using the basic data of the previous study (6), general and specific geographic and age differences between numbers of persons with 1 or more simple and compound cavities and restorations, teeth missing and to be extracted, are here reported. Emphasis is placed upon the differences in numbers of persons with these dental defects, and not upon the varying degrees of occurrence of each type of defect in individuals. The degree to which each dental defect occurs in these populations will be the subject of another report.

PROCEDURE

The mechanics of procurement of data have been described (6). The continental United States was divided into various regions according to place of birth of persons in this sample (*fig. 1*). The persons were arranged according to the number and per cent, by age groups for each region of birth (Tables I and II).

The number and per cent of persons with 1 or more simple cavities, compound cavities, simple restorations, compound restorations, teeth to be extracted and

TABLE I

Distribution of naval personnel examined in 1942 by age group and region of birth

AREA	AGE (YEARS)							
	All ages	17–19	20–24	25–29	30–34	35–39	40–44	45 and over
All regions	71,015	18,649	29,477	9,893	6,961	3,792	1,625	618
Continental United States	69,584	18,542	29,002	9,697	6,683	3,557	1,539	564
Abroad	1,431	107	475	196	278	235	86	54
Continental United States								
New England	6,014	1,545	2,603	862	528	278	134	64
Middle Atlantic	12,972	3,183	5,941	1,718	1,130	609	277	114
East North Central	7,474	1,673	2,762	1,253	906	551	251	78
West North Central	6,502	1,182	2,497	1,200	838	508	197	80
South Atlantic	11,116	3,718	4,610	1,284	856	407	183	58
East South Central	5,747	1,722	2,421	676	498	265	114	51
West South Central	10,332	2,814	4,024	1,486	1,133	565	231	79
Mountain	3,672	904	1,697	508	337	150	57	19
Pacific	5,755	1,801	2,447	710	457	224	95	21

TABLE II

Distribution of percentages of naval personnel examined in 1942 by region of birth within age groups

AREA	AGE (YEARS)							
	All ages	17–19	20–24	25–29	30–34	35–39	40–44	45 and over
All regions	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
Continental United States	97.98	99.43	98.39	98.02	96.01	93.80	94.71	91.26
Abroad	2.02	0.57	1.61	1.98	3.99	6.20	5.29	8.73
Continental United States	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
New England	8.64	8.33	8.98	8.89	7.90	7.82	8.71	11.35
Middle Atlantic	18.64	17.17	20.49	17.72	16.91	17.12	18.00	20.21
East North Central	10.74	9.02	9.52	12.92	13.56	15.49	16.31	13.83
West North Central	9.34	6.37	8.61	12.38	12.54	14.28	12.80	14.18
South Atlantic	15.98	20.05	15.89	13.24	12.81	11.44	11.89	10.28
East South Central	8.26	9.29	8.35	6.97	7.45	7.45	7.41	9.04
West South Central	14.85	15.18	13.87	15.32	16.95	15.88	15.01	14.00
Mountain	5.28	4.88	5.85	5.24	5.04	4.22	3.70	3.36
Pacific	8.27	9.71	8.44	7.32	6.84	6.30	6.17	3.72

teeth missing, were determined by regions of birth and successive age groups. The Chi Square test (7) was used to establish the significance of general differences between regions of birth (Table III), and between successive age groups (Table IV).

TABLE III

Distribution of dental defects according to region of birth

REGION OF BIRTH	NO. OF PERSONS EXAMINED	NUMBER AND PER CENT OF PERSONS OBSERVED WITH ONE OR MORE:											
		Simple restorations		Compound restorations		Simple cavities		Compound cavities		Teeth missing		Teeth to be extracted	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
New England	6,014	4,556	75.8 SP 75.7	3,743	62.3 SP 62.3	4,312	71.7 SP 71.4	2,810	46.8 SP 46.7	5,665	94.2 SP 94.2	725	12.1 SP 12.1
Middle Atlantic	12,972	10,084	77.8 SP 77.6	8,182	63.1 SP 63.1	9,177	70.7 SP 70.1	5,420	41.8 SP 41.6	12,347	95.2 SP 95.3	1,524	11.8 SP 11.8
East North Central	7,474	5,141	68.8 SP 68.2	4,237	56.7 SP 55.2	4,731	63.3 SP 65.5	3,042	40.7 SP 41.6	6,928	92.7 SP 92.7	772	10.4 SP 10.7
West North Central	6,502	4,113	63.3 SP 61.3	3,311	51.0 SP 47.8	3,937	60.6 SP 62.6	2,436	37.5 SP 38.1	5,775	88.8 SP 89.0	581	9.0 SP 9.0
South Atlantic	11,116	5,446	49.0 SP 50.9	3,638	32.2 SP 35.0	7,835	70.5 SP 68.7	3,449	31.0 SP 31.2	9,736	87.6 SP 87.2	1,647	14.9 SP 14.7
East South Central	5,747	2,541	44.3 SP 45.6	1,689	29.4 SP 30.9	3,922	68.2 SP 67.5	1,865	32.5 SP 32.4	4,929	85.8 SP 85.7	495	8.7 SP 8.7
West South Central	10,332	4,166	40.4 SP 40.2	2,906	28.2 SP 27.9	6,350	61.5 SP 61.7	3,086	29.9 SP 30.0	8,698	84.2 SP 84.0	675	6.6 SP 6.6
Mountain	3,672	2,035	55.5 SP 55.2	1,624	44.3 SP 44.3	2,384	64.9 SP 64.3	1,335	36.4 SP 36.1	3,172	86.4 SP 86.7	224	6.1 SP 6.2
Pacific	5,755	3,894	67.7 SP 68.5	3,293	63.7 SP 58.8	3,664	63.7 SP 62.1	2,320	40.4 SP 39.7	5,189	90.2 SP 90.3	290	5.1 SP 5.1

Abroad	1,431	1,042	72.9 SP 72.5	893	62.5 SP 59.1	770	53.8 SP 63.0	476	33.3 SP 38.1	1,303	91.1 SP 91.7	110	7.7 SP 9.6
Total	71,015	43,018	60.58	33,516	47.20	47,082	66.3	26,239	36.95	63,742	89.76	7,043	9.92
X^2 = (probability < 1%)		5,883+		5,714+		670+		946+		1,532+		749+	

Chi-square test of significance (x^2)

$$x^2 = \frac{(Fo_1 - Ft_1)^2}{Ft_1} + \frac{(Fo_2 - Ft_2)^2}{Ft_2} + \text{etc.}$$

Fo = observed number with defect.
Ft = theoretical number with defect.
Standard percentage by the direct method (SP)

$$SP = \frac{R_1Y_1 + R_2Y_2 \cdots R_7Y_7}{N}.$$

R_1 to R_7 = per cent with one or more type dental defect in age group for a single region of birth (use table 7).
Y_1 to Y_7 = total population in each age group (use table 1).
N = total number observed.

TABLE IV

Distribution of dental defects according to age

AGE GROUPS	NO. OF PERSONS EXAMINED	NUMBER AND PER CENT OF PERSONS WITH ONE OR MORE:											
		Simple restorations		Compound restorations		Simple cavities		Compound cavities		Teeth missing		Teeth to be extracted	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
17–19	18,649	9,061	48.6	5,864	31.4	14,074	75.5	7,084	38.0	17,551	94.1	1,985	11.9
20–24	29,477	18,311	62.1	13,817	46.9	21,253	72.1	11,483	39.0	25,585	86.8	3,092	10.4
25–29	9,893	6,764	68.4	5,802	58.6	5,863	59.3	3,636	36.8	8,650	87.4	964	9.7
30–34	6,961	4,990	71.7	4,455	64.0	3,468	49.8	2,333	33.5	6,305	90.6	612	8.7
35–39	3,792	2,616	69.0	2,412	63.6	1,592	42.0	1,103	29.1	3,515	92.7	239	6.3
40 and over	2,243	1,276	56.9	1,166	52.0	832	37.1	600	26.8	2,136	95.2	151	6.7
Total.....	71,015	43,018	60.58	33,516	47.20	47,082	66.30	26,239	36.95	63,742	89.76	7,043	9.92
X^2 = (probability < 1%)		1,888+		3,599+		670+		370+		837+		112+	

$$X^2 = \frac{(Fo_1 - Ft_1)^2}{Ft_1} + \frac{(Fo_2 - Ft_2)^2}{Ft_2} + \text{etc.}$$

Chi-square test of significance (X^2).
Fo = observed number with defects.
Ft = theoretical number with defects.

The standard error of the percentage (8) of those persons with each type dental defect here considered, was ascertained for each age group. Then by computing the standard error of the difference between the per cent of persons with each type of dental defect for each age group (9), the probable chance occurrence of these differences was established (Table V).

TABLE V

Observed percentages of persons with one or more dental defects by age groups (comparisons of age groups are to be made within each column, and are arranged in order of the size of the percentage)

SIMPLE RESTORATIONS		COMPOUND RESTORATIONS		SIMPLE CAVITIES		COMPOUND CAVITIES		TEETH MISSING		TEETH TO BE EXTRACTED	
Age	%	Age	%	Age	%	Age	%	Age	%	Age	%
		B				C		F			
30–34	71.7	30–34	64.0	17–19	75.5	20–24	39.0	40 & over	95.2	17–19	11.9
A		B				C D		F		H	
55–39	69.0	35–39	63.6	20–24	72.1	17–19	38.0	17–19	94.1	20–24	10.4
A						D				H I	
25–29	68.4	25–29	58.6	25–29	59.3	25–29	36.8	35–39	92.7	25–29	9.7
										I	
20–24	62.1	40 & over	52.0	30–34	49.8	30–34	33.5	30–34	90.6	30–34	8.7
						E		G		J	
40 & over	56.9	20–24	46.9	35–39	42.0	35–39	29.1	25–29	87.4	40 & over	6.7
						E		G		J	
17–19	48.6	17–19	31.4	40 & over	37.1	40 & over	26.8	20–24	86.8	35–39	6.3

Paired boldface letters indicate age groups between which there are no significant differences, i.e. probability <1%.

$$\text{Test of significance of difference between age groups} = \frac{\text{Difference}}{\text{standard error of difference}}$$

$$= \frac{p_1 - p_2}{\sqrt{p_1 \times q_1/N_1 + p_2 \times q_2/N_2}}$$

p = per cent of persons observed with type dental defect.
q = per cent of persons observed without type dental defect.
N = number of persons examined in each age group.

The incidence of each type dental defect for each region of birth was standardized by the direct method (10) (Table III), thereby reducing the variables which might be introduced by the dissimilar totals of persons observed in each age group for each region of birth. Using these percentages, the reality of differences between regions of birth was computed by finding the standard error of the difference between proportions (9) (Table VI).

Actual percentages of combined regional and age distribution of persons with one or more of a given type of dental defect were also determined (table VII).

TABLE VI

Standard percentages of persons with one or more specific dental defects by region of birth (comparison of regions of birth are to be made within each column, and regions are arranged in order of the size of the percentage)

SIMPLE RESTORATIONS		COMPOUND RESTORATONS		SIMPLE CAVITIES		COMPOUND CAVITIES		TEETH MISSING		TEETH TO BE EXTRACTED	
Region	%	Region	%	Region	%	Region	%	Region	%	Region	%
		B		**E**							
MA	77.6	MA	63.1	NE	71.4	NE	46.7	MA	95.3	SA	14.7
		B C		**E**		**R**				**3**	
NE	75.7	NE	62.3	MA	70.1	MA	41.6	NE	94.2	NE	12.1
		C D		**F**		**R S**		**X**		**3**	
ABR	72.5	ABR	59.1	SA	68.7	ENC	41.6	ENC	92.7	MA	11.8
A		**D**		**F**		**S T**		**X Y**		**4**	
PAC	68.5	PAC	58.8	ESC	67.5	PAC	39.7	ABR	91.7	ENC	10.7
A				**G H**		**T**		**Y**		**4 5 6**	
ENC	68.2	ENC	55.2	ENC	65.5	ABR	38.1	PAC	90.3	ABR	9.6
				G I J K		**U**				**5 7**	
WNC	61.3	WNC	47.8	MT	64.3	WNC	38.1	WNC	89.0	WNC	9.0
				H I L M N		**U**		**Z**		**6 7**	
MT	55.2	MT	44.3	ABR	63.0	MT	36.1	SA	87.2	ESC	8.7
				J M O P		**V**		**Z 2**		**8**	
SA	50.9	SA	35.0	WNC	62.6	ESC	32.4	MT	86.7	WSC	6.6
				K N P Q		**V W**		**2**		**8 9**	
ESC	45.6	ESC	30.9	PAC	62.1	SA	31.2	ESC	85.7	MT	6.2
				L O Q		**W**				**9**	
WSC	40.2	WSC	27.9	WSC	61.7	WSC	30.0	WSC	84.0	PAC	5.1

Paired **bold face letters** or **numbers** indicate regions between which there are no significant differences, i.e. probability <1%.

$$\text{Test of significance of difference between regions of birth} = \frac{\text{Difference}}{\text{standard error of difference}}$$

$$= \frac{p_1 - p_2}{\sqrt{p_1 \times q_1/N_1 + p_2 \times q_2/N_2}}$$

p = per cent of persons observed with type dental defect.
q = per cent of persons observed without type dental defect.
N = number of persons examined in region.

MA = Middle Atlantic, NE = New England, PAC = Pacific, ENC = East North Central, WNC = West North Central, MT = Mountain, SA = South Atlantic, ESC = East South Central, WSC = West South Central, ABR = Abroad.

RESULTS AND COMMENT

Significant general regional and age differences were found between persons with 1 or more simple and compound restorations and cavities, teeth missing, and teeth to be extracted (Tables III and IV).

When the numbers of persons in different age groups with one type of dental defect were compared with each other, significant differences were found for all

type dental defects, except as noted (Table V). Generaly, when age groups of persons with 1 or more simple and compound restorations occupied high positions in the percentage table, these same age groups were in lower positions with respect to simple and compound cavities. This was not true for age group 40 and over, which group occupied lower positions in the table for persons with these defects. Age group positions of persons with one or more simple and compound cavities and teeth to be extracted were similar.

When the standard percentages of persons with 1 or more of the type dental defect in the different regions of birth were compared with each other, significant differences were found between regions for all type dental defects, except as noted (table VI). Where insignificant differences existed between persons born in different regions with regard to 1 type dental defect, the position of these regions in the table were found to be similar for that defect. Generally fewer persons born in the West South Central and East South Central regions showed dental defects than those born in the New England and Middle Atlantic regions. The same results were generally found in most age groups in these regions when the extremes of the actual percentages were analyzed in more detail (Table VII).

The number of persons with simple and compound cavities for all regions of birth, diminishes to a varying degree as age increases. This causes a shift in the regional, positional relationship in the percentage table (Table VII). The West South Central and East South Central regions move irregularly to a higher level, and the New England and Middle Atlantic regions to a lower position as age advances. This change in position is more pronounced for persons with simple cavities born in the East South Central region and for those with compound cavities born in the West South Central region, than for persons born in other regions with these defects. However, since most age groups in the West South Central and, to a lesser extent, the East South Central regions of birth show fewer persons with simple and compound restorations and teeth missing than other regions, and an irregular, relative increase is noted in the percentage table for these persons requiring extractions as age increases, it might indicate that the carious process is slower or delayed, and that teeth may require extractions for reasons other than dental caries.

For most age groups, the South Atlantic region consistently had most persons with 1 or more teeth to be extracted (Table VII). The standard percentages of each type dental defect in the South Atlantic region (Table VI) were relatively low with respect to simple and compound restorations, compound cavities and teeth missing, higher for simple cavities, and highest for teeth to be extracted.

The East South Central region of birth is inconsistent with its general position in the percentage table (Table VI) for persons with simple cavities. For most other defects its position is rather low in comparison to other regions.

The Pacific region of birth is inconsistent with its general position in the percentage table (Table VI) for persons with 1 or more teeth to be extracted and simple cavities, being generally lowest for these defects.

The pattern of age group distribution of persons with 1 or more of the type dental defect (Table V), follows general impressions gained in a clinical dental

TABLE VII

Observed percentage of persons with one or more specific dental defects by region of birth within age groups (comparisons of regions of birth within age groups are to be made within each horizontal line, and the regions of birth are arranged horizontally in order of the size of the percentage

	AGE GROUP 17–19																					
Simple restorations	NE	72.1	MA	71.6	ABR	65.5	PAC	58.7	ENC	57.1	WNC	49.3	MT	42.2	SA	36.3	ESC	30.1	WSC	27.0		
Compound restorations	NE	54.0	MA	49.9	ABR	47.7	PAC	43.4	ENC	37.7	WNC	29.6	MT	29.2	SA	19.7	WSC	14.2	ESC	14.0		
Simple cavities	NE	83.3	MA	82.3	SA	77.4	ABR	76.6	ENC	76.3	ESC	73.6	MT	70.5	WNC	70.5	PAC	70.2	WSC	68.5		
Compound cavities	NE	52.3	MA	46.9	ENC	44.9	ABR	44.0	PAC	43.3	WNC	39.5	MT	34.6	ESC	32.8	SA	28.8	WSC	28.1		
Teeth missing	MA	97.6	NE	96.3	ENC	96.0	ABR	95.3	WNC	94.1	SA	93.2	PAC	93.1	MT	92.5	WSC	92.2	ESC	90.8		
Teeth to be extracted	NE	15.7	SA	14.7	ABR	14.1	MA	13.7	ENC	11.6	WNC	9.0	ESC	8.1	MT	6.7	WSC	6.0	PAC	4.6		
	AGE GROUP 20–24																					
Simple restorations	MA	79.9	NE	79.2	ABR	76.9	ENC	71.0	PAC	70.0	WNC	61.2	MT	58.6	SA	50.8	ESC	42.6	WSC	39.3		
Compound restorations	MA	64.6	NE	64.0	ABR	61.5	PAC	58.6	ENC	57.1	WNC	47.6	MT	44.5	SA	32.5	ESC	26.9	WSC	23.3		
Simple cavities	MA	77.7	NE	76.9	SA	74.1	ESC	73.5	ENC	71.4	MT	68.6	PAC	67.7	ABR	66.5	WNC	66.4	WSC	66.4		
Compound cavities	NE	49.2	ENC	44.0	MA	43.6	ABR	41.9	PAC	41.5	WNC	39.5	MT	38.8	ESC	33.0	SA	32.2	WSC	31.7		
Teeth missing	MA	94.1	NE	93.8	ABR	91.2	ENC	90.6	PAC	86.2	WNC	82.2	MT	84.3	SA	83.4	ESC	80.2	WSC	78.7		
Teeth to be extracted	SA	16.4	MA	12.4	NE	12.0	ENC	12.0	WNC	9.2	ESC	9.0	ABR	8.7	WSC	6.4	MT	6.0	PAC	5.1		
	AGE GROUP 25–29																					
Simple restorations	MA	82.0	ENC	76.3	NE	76.1	ABR	74.5	WNC	72.5	PAC	71.2	MT	62.1	SA	61.0	ESC	58.6	WSC	49.2		
Compound restorations	MA	73.5	PAC	67.9	ENC	67.6	ABR	67.4	NE	66.6	WNC	61.4	MT	54.8	SA	46.2	ESC	44.1	WSC	40.5		
Simple cavities	NE	64.6	SA	62.2	ESC	61.0	WNC	59.4	MA	58.8	MT	57.9	PAC	57.6	ENC	57.4	WSC	56.7	ABR	55.1		
Compound cavities	NE	42.4	PAC	41.1	WNC	39.0	MA	38.6	ENC	38.4	MT	35.3	SA	33.7	ESC	33.4	WSC	31.9	ABR	31.7		
Teeth missing	MA	94.9	NE	91.7	ENC	90.5	PAC	90.4	ABR	88.3	WNC	87.3	ESC	84.5	MT	83.7	SA	82.2	WSC	79.5		
Teeth to be extracted	SA	14.2	MA	11.5	NE	10.6	WNC	10.0	ESC	9.2	ENC	9.1	ABR	7.7	WSC	7.3	PAC	6.7	MT	5.8		

	AGE GROUP 30–34																					
Simple restorations	PAC	83.4	MA	81.8	NE	75.8	ENC	75.7	WNC	74.4	ABR	72.7	SA	67.5	ESC	66.7	MT	65.0	WSC	57.2		
Compound restorations	PAC	79.9	MA	72.9	NE	72.6	ENC	69.1	WNC	67.3	ABR	63.0	MT	60.6	ESC	55.3	SA	55.2	WSC	50.2		
Simple cavities	MT	55.5	SA	53.5	WNC	52.1	ESC	51.2	WSC	50.1	NE	49.6	MA	47.4	PAC	47.2	ENC	46.9	ABR	45.0		
Compound cavities	ENC	37.1	NE	36.8	MT	36.2	WNC	35.8	PAC	34.4	MA	34.3	ESC	32.4	SA	31.5	WSC	29.5	ABR	28.1		
Teeth missing	PAC	95.0	MA	94.1	ENC	93.8	NE	93.2	WNC	90.2	SA	90.2	ESC	90.0	ABR	86.7	WSC	85.5	MT	83.1		
Teeth to be extracted	SA	12.7	NE	10.1	ESC	9.5	ENC	8.9	WNC	8.9	MA	8.7	WSC	7.6	ABR	7.6	MT	7.2	PAC	4.6		
	AGE GROUP 35–39																					
Simple restorations	MA	79.5	ABR	74.9	NE	74.1	PAC	73.3	ESC	71.7	ENC	71.2	WNC	66.5	SA	66.1	MT	60.7	WSC	54.2		
Compound restorations	MA	73.3	ABR	71.1	PAC	70.1	ENC	69.4	NE	67.3	ESC	61.6	WNC	61.1	MT	60.0	SA	56.8	WSC	49.4		
Simple cavities	ESC	51.7	MT	46.7	SA	45.7	NE	45.0	MA	40.9	WNC	40.9	WSC	40.5	ABR	40.0	ENC	39.4	PAC	34.4		
Compound cavities	NE	38.2	ENC	30.9	MA	30.1	MT	30.0	WNC	29.6	SA	29.5	ESC	29.5	WSC	25.0	ABR	23.9	PAC	23.3		
Teeth missing	PAC	96.4	MA	95.1	NE	94.6	ENC	94.4	ABR	93.6	ESC	93.6	WNC	92.1	SA	90.7	WSC	88.7	MT	87.3		
Teeth to be extracted	SA	7.9	ESC	7.2	WSC	7.1	WNC	7.1	MA	6.1	ABR	6.0	NE	5.8	PAC	5.4	ENC	5.3	MT	2.7		
	AGE GROUP 40–44																					
Simple restorations	MA	67.5	PAC	67.4	WNC	64.7	NE	62.7	ENC	62.2	ABR	60.5	SA	58.5	MT	50.9	ESC	46.5	WSC	46.4		
Compound restorations	PAC	67.4	MA	63.6	WNC	59.9	ENC	56.4	NE	51.5	SA	51.4	ABR	48.9	MT	47.4	ESC	45.7	WSC	43.8		
Simple cavities	MT	50.9	ESC	49.1	NE	46.3	SA	45.9	ENC	40.2	MA	39.0	PAC	38.5	WSC	38.1	WNC	34.0	ABR	32.6		
Compound cavities	NE	33.6	SA	33.4	PAC	32.7	ENC	29.5	WSC	28.5	MA	28.2	MT	28.1	ESC	24.6	WNC	23.4	ABR	22.1		
Teeth missing	ENC	96.8	ABR	96.5	MA	96.4	SA	95.6	WNC	95.4	NE	94.8	PAC	94.7	ESC	93.9	WSC	92.2	MT	91.2		
Teeth to be extracted	SA	12.1	MT	8.8	ENC	8.0	ESC	7.9	WSC	7.4	MA	7.3	NE	6.8	PAC	5.3	WNC	4.6	ABR	3.5		
	AGE GROUP 45 AND OVER																					
Simple restorations	WNC	58.8	ABR	57.5	NE	54.7	PAC	52.4	MA	51.8	ENC	50.0	WSC	46.9	ESC	43.2	SA	39.7	MT	36.9		
Compound restorations	ABR	63.0	WNC	58.9	PAC	52.4	NE	48.5	ENC	46.2	MA	45.7	MT	36.9	SA	34.5	ESC	33.4	WSC	33.0		
Simple cavities	PAC	33.4	MA	31.6	ABR	31.5	SA	29.3	WSC	29.1	NE	28.1	ESC	27.4	ENC	25.6	WNC	25.0	MT	15.8		
Compound cavities	ABR	27.8	WNC	26.3	MA	24.6	ENC	23.1	WSC	22.8	NE	20.4	PAC	19.1	SA	18.7	MT	15.8	ESC	11.8		
Teeth missing	PAC	100	MA	97.4	NE	96.9	WNC	96.3	ESC	96.1	ENC	94.9	SA	94.8	MT	94.7	ABR	94.4	WSC	92.4		
Teeth to be extracted	WNC	8.8	SA	6.9	MA	6.2	ESC	5.9	MT	5.3	ENC	5.2	NE	4.7	WSC	2.6	ABR	1.9	PAC	.0		

practice. In the age group of those 40 and over, the relatively low position for simple cavities and restorations and compound cavities and restorations may be due to the increase of persons with teeth missing at this age, as seen in the percentage table (Table V).

The 17–19 age group stands high in the percentage column for persons with 1 or more teeth missing, probably because of incomplete eruption of all third molars at this age, rather than loss through dental caries as suggested by other writers (1, 5) (Table V). If dental caries were the important etiologic factor for persons with teeth missing in this age group, then the relatively low position of age groups 20–24 and 25–29 could not be explained. In the following report this problem will be discussed in greater detail.

The inconsistency of the standard percentages for different dental defects in the South Atlantic region (Table VI) might suggest an insufficiency of dental care. Since teeth to be extracted are only so indicated on the dental record without registration as to class of carious lesions or reason for such extractions, the relatively low rate for compound cavities in this region may be partially explained. With so many persons in this region with teeth to be extracted and simple cavities, and fewer with simple and compound restorations, it seems logical to assume an insufficiency of dental attention.

Explanation of other inconsistencies which appear in the regional tables for persons with some of the type dental defects, may have to await analysis of data on rate occurrence of these dental defects in persons with them.

SUMMARY

1. The number of persons showing simple and compound cavities and restorations, teeth missing and teeth to be extracted, varied significantly according to their regions of birth in the United States. Generally speaking, more persons born in the New England and Middle Atlantic regions were dentally defective than those born in the West South Central and East South Central regions, particularly in the younger age groups. Examples of the high and low extremes in regional standard percentages of persons with 1 or more of the type dental defect are: simple restorations for Middle Atlantic 77.6 % and for West South Central 40.2%; compound restorations for Middle Atlantic 63.1% and for West South Central 27.9%; simple cavities for New England 71.4 % and for West South Central 61.7%; compound cavities for New England 46.7% and for West South Central 30.0%; teeth missing for Middle Atlantic 95.3% and for West South Central 84.0%; and teeth to be extracted for South Atlantic 14.7% and for Pacific 5.1%.

2. The percentage of persons in the older age groups with restorations (fillings) and missing teeth was greater, and with cavities and teeth to be extracted was less than in the younger age groups. These results agree with the impressions gained in a dental practice. Examples of the high and low extremes in age group percentages of persons with 1 or more of the type dental defect are: simple restorations for 30–34, 71.7% and for 17–19, 48.6%; compound restorations for 30–34, 64.0% and for 17–19, 31.4%; simple cavities for 17–19, 75.5% and for 40

and over, 37.1%; compound cavities for 20–24, 39.0% and for 40 and over, 26.8%; teeth missing for 40 and over, 95.2% and for 20–24, 86.8%; and teeth to be extracted for 17–19, 11.9% and for 35–39, 6.3%.

The authors are indebted to Captain F. Lang, (MC), USN, Commander F. E. Linder, H(S), USNR, and the Division of Vital Statistics of the Bureau of Medicine and Surgery, Navy Department, for their assistance in applying some of the statistical methods to these results, and to James E. Horlander, PhM2c, V6, USNR, who assisted in the computation of the data.

REFERENCES

1. Ferguson, R. A., Some observations on diet and dental disease, *J. A. D. A.*, **22:** 392–401, 1935.
2. Senn, W. W., Incidence of dental caries among aviation cadets, *The Mil. Surg.*, **93:** 461–464, 1943.
3. Schlack, C. A., Dental caries experience at initial examination and the incidence of new carious lesions after a one year interval in 1047 officers and men of the United States Navy, *U. S. Nav. Med. Bul.*, **38:** 90–95, 1940.
3a. Schlack, C. A., Rate of increase of dental caries in 707 officers and men aboard an American man of war, *J. D. Res.*, **20:** 477–481, 1941.
4. Hollander, F., and Dunning, J. M., A study by age and sex of the incidence of dental caries in over 12,000 persons, *J. D. Res.*, **18:** 43–59, 1939.
5. East, B. R., Some epidemiological aspects of tooth decay, *Amer. J. Pub. Health*, **32:** 1242–1250, 1942.
6. Restarski, J. S., Schlack, C. A., and Dochterman, E. F., Dental status of 71,015 naval personnel at first examination in 1942, Research Project X-131, Report No. 1, *Naval Medical Research Institute* 26 Nov. 1945.
7. Hill, A. Bradford, *Principles of Medical Statistics* (London, The Lancet Limited: 1945) pp. 78–96.
8. *Idem*, *Ibid.*, pp. 58–67.
9. *Idem*, *Ibid.*, pp. 68–77.
10. Linder, F. E., and Grave, R. D., Vital statistic rates in the United States, 1900–1940 (The U. S. Government Printing Office: 1943) pp. 66–69.

CLINICAL AND BACTERIOLOGICAL STUDIES OF THE USE OF A FLUORIDE DENTIFRICE[1]

EDWARD O. SHANER, D.D.S.

Washington, D. C.

AND

R. REED SMITH, D.D.S.

Springfield, Mo.

The mechanism by which fluorine acts to reduce dental caries is not yet clearly understood. It has been shown that fluorine has no marked bactericidal powers, when tested in vitro (1). However, fluorine may have the ability to restrict the action of the cellular enzymes responsible for the breakdown of sugars to acids. Phosphatase is greatly inhibited, thereby blocking the conversion of phosphoglycerinic acid to phosphopyruvic acid and thence to pyruvic and lactic acid. Other enzymes as salivary amylase, lipase, and proteolytic and diastatic enzymes are not affected by the fluoride ion (2). Although these studies have been conducted chiefly in animal tissues and yeast, there is little doubt that the enzymic mechanisms of bacterial fermentation are similarly affected.

Bibby reported that fluorine, added to cultures of oral acidogenic bacteria, inhibited acid production. The fluorine was effective at a concentration of 1 part per million, and markedly so, at concentrations of 250 and 1000 p.p.m. Fluorosed and fluorine treated enamel and dentin, also reduced acid production in bacterial cultures (1). Therefore, it is not unreasonable to conclude that some similar action would occur, when fluorine came into the presence of acidogenic organisms in the human mouth. Atkins stated that a reduction of cell counts can be achieved by the routine use of a fluoride mouthwash (3).

To study the effect of the daily use of sodium fluoride on oral acidogenic bacteria, an experiment embracing the use of a fluoride dentifrice was postulated. Preliminary work was done on a series of guinea pigs, to determine the gross toxic manifestations of the ingestion of lethal doses of the fluoride. It was found, that double the accepted lethal dose of sodium or potassium fluoride was well tolerated and did not produce death, when buffered with calcium carbonate, or calcium carbonate combined with magnesium oxide (4). The conversion of sodium fluoride to calcium fluoride, in the stomach of the animal, drastically reduced the toxicity of the fluoride, since calcium fluoride is practically insoluble and therefore only slightly assimilated. Guinea pigs, fed similar doses of sodium fluoride or potassium fluoride, unbuffered by calcium carbonate or magnesium oxide, died within 1 hour. The premise was forwarded, that a dentifrice contain-

[1] Received for publication March 26, 1946.

ing sodium fluoride and calcium carbonate would be a convenient method of topical fluorine therapy, as the fluoride would be free to act in the mouth, yet be changed to the highly insoluble calcium fluoride if it were to be ingested accidentally.

EXPERIMENTAL PROCEDURE

Twelve subjects were issued a dentifrice containing 0.1% sodium fluoride, and the teeth were brushed twice per day, directly in the morning, and at the end of the working day. This procedure was done under supervision in the dental office. Approximately 0.5 gms. of powder was used at each brushing, which was timed to last, not less than 3 minutes. The mouth was thoroughly rinsed after each brushing, for a period of 1 minute. Each subject, therefore, applied 1 gm. of dentifrice to his teeth per day. This contained 0.001 gm. of fluoride. After 30 days, it was decided to increase the amount of fluoride in the dentifrice to 0.5% and to expand the group of subjects. Fifty individuals were examined and their caries and gingival conditions were charted. Special note was made of any sensitive gingival areas, which were painful when mechanically or thermally irritated, as fluorine is known to have an obtunding effect when applied topically (5). The patients were instructed in the proper use of the dentifrice and given orders to report to the dental office weekly for observation. At the end of the 30 days, a check was made on all patients using the experimental dentifrice. The results were as follows: 1. In all but 5 cases, the gingiva were normal and healthy and possessed good firm tone. The 5 exceptions were individuals with marked periodontal disease accompanied by gingivitis. These showed no changes from their original condition. 2. Cervical areas on teeth, charted as sensitive to thermal changes, air, and the toothbrush, were somewhat improved. 3. No clinical symptoms of fluorine toxicosis were discernible.

The second phase of the investigation centered on the effect of this dentifrice on acidogenic oral flora, particularly the lactobacilli. Experimental work has shown that the lactobacillus determination of the saliva forms a fairly reliable index as to the caries activity (6). When the count is reduced to a low level, decay ceases or is greatly arrested. An agent which reduces the lactobacillus count, or prevents the organisms from converting the sugars to acids, should produce a decrease in dental caries.

The procedure for the collection of saliva specimens followed that recommended by the Missouri State Board of Health. The method is as follows: 1. A paraffin square is placed in the mouth and chewed vigorously, shifting it from one side of the mouth to the other so all teeth are used. 2. Saliva is collected for exactly 3 minutes. The paraffin is discarded. 3. Saliva specimen is expectorated into sterile specimen bottle. The top is screwed on tightly, care being taken to avoid touching inside of the top. 4. Specimen and record card are mailed to laboratory the same day the specimen is taken.

Forty-seven new subjects were selected to participate in this phase of the investigation. A thorough oral examination with mouth mirror and explorer was given each patient. All missing teeth, filled teeth, and all carious areas were

TABLE I

CASE NO.	AGE	RACE	RESIDENCE 1ST 8 YRS.	ERUPTED PERMANENT TEETH	DECAYED PERMANENT TEETH	LOST PERMANENT TEETH	FILLED PERMANENT TEETH	NO. SENSITIVE TEETH	NO. TEETH DESENSITIZED	LACTOBACILLUS COUNTS								INCREASE OR DECREASE
										First series				Second series				
										1	2	3	Average	1	2	3	Average	
"A" experimental group																		
1	21	W	Pa.	27	1	2	20	0	0	249,000	500,000+	*	374,500	117,000	2,000	500	39,890	−334,610
2	26	W	Va.	27	2	5	7	3	2	36,000	500,000	39,000	191,000	18,000	*	231,000	83,000	−112,000
3	47	W	Fla.	27	0	5	8	0	0	−100	*	6,000	3,500	3,000	*	3,000	3,000	−500
4	30	W	Ala.	31	0	1	9	5	2	−100	*	−100	−100	−100	−100	−100	−100	same
5	20	W	Wash.	30	2	2	8	0	0	3,000	−100	−100	1,060	1,750	2,500	2,500	2,250	+1,190
6	26	N	Utah	25	8	7	2	2	1	100	100	*	100	500	250	−100	283	+183
7	24	W	Ky.	30	0	0	10	6	6	5,000	500,000+	*	168,000	12,000	4,500	*	8,250	−159,750
8	22	W	N. J.	29	6	3	0	6	0	384,000	500,000+	390,000	421,300	3,600	*	*	3,600	−417,700
9	19	W	W. Va.	27	1	5	5	12	3	60,000	500,000+	100	186,700	−100	−100	9,000	3,066	−183,634
10	24	W	W. Va.	27	0	5	1	6	0	45,000	500,000+	150,000	252,000	−100	51,000	21,000	24,033	−207,967
11	27	W	Tenn.	30	7	2	1	3	1	24,500	330,000	279,000	112,160	93,000	45,000	*	69,000	−43,160
12	37	W	Kan.	29	8	3	8	0	0	−100	*	−100	−100	*	*	−100	−100	same
13	36	W	Wash.	26	0	6	5	4	3	100	100	12,000	4,070	−100	−100	*	−100	−3,970
14	22	W	Tex.	22	8	10	8	1	0	27,000	*	30,000	28,500	267,000	60,000	−100	109,033	+80,533
15	21	W	N. Y.	32	12	0	0	0	0	6,000	6,000	*	6,000	−100	−100	*	−100	−5,900
"B" control group																		
16	33	W	Miss.	28	0	4	0	0	0	258,000	†	750	150,375	258,000	42,000		150,000	−375
17	23	W	W. Va.	24	2	8	3	1	0	48,000	90,000	177,000	105,000	6,000	140,000	112,000	104,000	−1,000
18	30	W	Okla.	29	2	3	6	0	0	−100	4,000	500,000+	168,000	6,000	500,000+	2,950	169,650	+1,650
19	27	W	Minn.	28	0	4	18	0	0	500	5,750	750	2,333	−100	8,000	1,750	3,283	+950
20	20	W	Ind.	27	3	5	3	4	0	−100	500,000+	500,000+	333,360	168,000	500,000+	500,000+	389,333	+55,973
21	46	W	Cal.	30	0	2	3	0	0	458,000	−100	−100	152,700	−100	250,000	390,000	213,360	+60,660
22	37	W	Mex.	27	3	5	0	2	0	−100	−100	−100	−100	1,500	750	−100	783	+683
23	46	W	N. Y.	27	0	5	9	1	0	*	18,000	13,500	15,750	*	21,000	1,050	11,025	−4,725
24	25	W	Cal.	31	0	1	16	4	0	†	750	750	750	−100	750	−100	316	−434
25	39	W	Ark.	23	2	11	0	2	0	411,000	108,000	42,000	187,000	75,000	83,000	250,000	196,000	+9,000
26	43	W	Mex.	28	0	4	0	0	0	110,000	100,000	*	105,000	114,000	*	190,000	152,000	+47,000
27	42	N	Wyom.	28	2	4	1	6	0	−100	†	500,000+	250,000	6,000	375,000	†	195,000	−55,000
28	18	W	Ohio	24	0	8	24	0	0	−100	4,750	500,000+	168,280	33,000	500,000+	1,250	178,083	+9,833
29	24	W	Wisc.	22	1	10	5	0	0	−100	500,000+	500,000+	333,366	−100	425,000	384,000	269,700	−63,666
30	27	W	Ohio	28	3	0	9	2	0	12,000	13,250	12,000	12,416	10,000	18,500	15,250	14,583	+2,168

−100 = below 100; 500,000+ = over 500,000.
* Overgrown with yeast.
† Unsatisfactory sample submitted.

TABLE II

Group "C" lactobacillus counts

CASE NO.	FIRST SERIES				SECOND SERIES				INCREASE OR DECREASE
	1	2	3	Average	1	2	3	Average	
31	450	500,000+	†	250,225	7,000	500,000+	15,000	174,000	−76,225
32	2,100	1,000	500,000+	167,700	12,000	500,000+		256,000	+89,300
33	500,000+	500,000+	500,000+	500,000+	2,200	1,000		1,600	−498,400
34	2,500	*	1,000	1,750	800	1,350	775	975	−775
35	800	500,000+	500,000+	333,600	6,800	2,400		4,600	−329,000
36	200	700	150	350	5,800	3,200	4,500	4,500	+4,150
37	31,200	500,000+	500,000+	343,733	51,200	15,000		33,100	−310,633
38	*	850	3,250	2,050	16,000	40,000		28,000	+25,950
39	†	65,000	84,500	74,750	2,000	8,600	6,000	5,533	−59,217
40	*	9,250	19,500	14,375	*	9,200	†	9,200	−51,750
41	3,500	*	1,550	2,525	*	20,000		20,000	+17,475
42	500,000+	500,000+		500,000+	500,000+	500,000+	500,000+	500,000+	same
43	500,000+	500,000+	500,000+	500,000+	290,000	500,000+		395,000	−105,000
44	500,000+	500,000+	2,200	334,066	4,000	7,600	6,100	5,900	−328,166
45	52,000	50,000	50,000	51,333	*	5,150	2,600	3,875	−47,458
46	3,000	70,000	1,200	24,733	6,500	3,650	2,850	4,333	−20,400
47	500,000+	*	500,000+	500,000+	27,300	3,100		15,200	−484,850

−100 = below 100; 500,000+ = over 500,000.

* Overgrown with yeast.

† Unsatisfactory specimen submitted to laboratory.

carefully charted. Condition of the gingival tissues, and the presence of any sensitive cervical areas on the teeth were also noted. Saliva samples were then obtained from each patient for 3 successive days. These samples were taken the first thing in the morning, under supervision in the dental office. Saliva samples were sent to the laboratories of the Missouri State Board of Health for lactobacillus determinations.

The subjects in group "A" were given a prophylaxis and were issued the experimental dentifrice. The subjects in group "B" (control group) were treated similarly, and were issued a dentifrice identical to that given group "A" except that it was fluorine free. Group "C" subjects were issued the experimental dentifrice, but they were not given a prophylaxis. The reason for giving groups "A" and "B" a prophylaxis was to start each subject with a uniformly clean mouth, and to teach each individual the desired brushing method, and to inspire the subject to cooperate with the dentist. Group "C" was observed for the effects of the dentifrice alone, without any change in the mouth or the oral hygiene of the patient.

Four weeks later, saliva samples were again procured from the subjects of all 3 groups, and the lactobacillus counts were determined. The results are shown in Tables I and II. In some instances a full series of lactobacillus counts were not obtained because the patients became temporarily unavailable, or the saliva samples leaked from the bottles during transit. In other instances, cultures were overgrown with yeast. The diet for all 3 groups was identical except as to the quantity of food consumed. The meals were high in carbohydrate and low in protein and fat. The intake of sugar and sweets was low. All of the subjects were hospital patients[2] and followed daily routines which were nearly identical.

The use of the fluoride dentifrice was extended to include over 200 patients. It was dispensed especially to the new admissions to the hospital, who upon examination in the dental office showed evidence of a high rate of caries susceptibility, in an effort to reduce the rate of tooth decay in these persons.

The patients using the fluoride dentifrice reported monthly to the dental office for observation. After 12 months each individual received a thorough oral examination. New caries were charted and sensitive cervical areas were again noted. Observation was continued for another 3 months.

DISCUSSION

Of the subjects in groups "A" and "C" which showed well correlated counts, 10 (Nos. 1, 10, 11, 15, 33, 37, 39, 44, 45, and 47) can be considered as showing a significant drop in lactobacillus counts following the use of the fluoride dentifrice. Two (Nos. 14 and 38) showed slight increase. Six individuals (Nos. 3, 4, 12, 13, 34, and 42) remained approximately the same. The lactobacillus counts in the remaining cases were inconsistent. This may be attributed to technical difficulties or lack of proper cooperation by the patient.

Well correlated counts in group "B" showed an increase in 4 subjects (Nos. 18, 19, 25, and 30) and a decrease in one (No. 29) and no noteworthy change in 6

[2] U. S. Medical Center, Springfield, Mo.

(Nos. 17, 20, 22, 23, 24, and 26). The counts in the remaining instances were considered inconsistent.

The dentifrice was used by patients for 15 months. In no case was there any irritation of the gingivae, nor was there any pathology which could be traced to the use of the fluoride dentifrice. No evidence of fluorine toxicosis was noted at any time.

This study was designed to throw light on a means of fluorine therapy which has received much speculation. It indicates that the daily application of minute amounts of fluoride to the teeth can produce a reduction in the count of acidogenic organisms in the saliva of humans. The experiment was conducted under close supervision and surveyance in a controlled group of adult subjects. Further studies on a larger group of subjects are in order.

Acknowledgement is made to Dr. A. E. Murphy of the Missouri State Board of Health for providing the facilities for obtaining the bacteriological counts on saliva samples.

REFERENCES TO LITERATURE

1. Bibby, B. G., The Effect of Fluorine on Mouth Bacteria, *J. D. R.*, **19**: 391, 1940.
2. Sollman, *Textbook of Pharmacology;* 6th Ed. (Philadelphia, W. B. Saunders Co.: 1942) pp. 843.
3. Atkins, P. A., Practical Caries Control, *J. A. D. A.*, **31**: 353, 1944.
4. Smith, R. R., and Shaner, E. O., Effects of Buffered Lethal Doses of Fluoride on Guinea Pigs, *J. A. D. A.*, **31**: 1483, 1944.
5. Bibby, B. G., and Hoyt, Wm. H., Use of Sodium Fluoride to Desensitize Dentin, *J. A. D. A.*, **30**: 1372, 1943.
6. Hadley, F. P., Quantitative Method of Estimating Bacillus Acidophilus in Saliva, *J. D. Res.*, **13**: 415, 1943.

DECAY OF THE DECIDUOUS TEETH OF FORMOSA CHINESE. AN ANALYTICAL SUMMARY AND INTERPRETATION OF CERTAIN STATISTICS PUBLISHED BY MARUYAMA[1]

V. O. HURME, D.M.D.

From the Department of Operative Dentistry, College of Dentistry, University of Illinois, Chicago, Ill.

At present there are exceedingly inadequate data available to occidental stomatologists who may be interested in the endemiology of dental diseases in the Far East, particularly in China, Korea and Japan. The best known reports in the English literature dealing with dental decay in this area are those written by Agnew (2), Anderson (3) and Montelius (11). Of these, only Anderson provides data on necrosis involving the deciduous teeth.

The lengthy report by Maruyama (9) has never been reviewed in American dental literature, which is quite understandable in view of the linguistic difficulties involved and the general inaccessibility of Formosan journals. This is most unfortunate from the standpoint of clinical pathology for several reasons. To one who has read the writings of Agnew (1), Ballantine (4) and others who have described the economic and social status of the Chinese masses, it is quite evident that the natives of Formosa and other Asiatic territories furnish the medical scientist an opportunity to study the health conditions of large population groups which do not enjoy the benefits of various public health measures, such as sanitation, vaccination, free medical care for the poor, etc. While these measures are generally taken for granted in most Western communities today, they are carried out to such extremely variable degrees in different localities that they frequently introduce numerous immeasurable variables for consideration by the student of human biology. It is difficult to determine the relative importance of a multitude of possible etiologic agents unless one can compare the data obtained for population groups living under complex environmental conditions with similar data obtained for comparable groups living under lesser degrees of artificial interference. One may say that as a general rule the endemiology and epidemiology of the less modernized peoples should yield meaningful diagnostic clues more readily than similar studies of the supposedly more "civilized" groups of mankind.

The investigation reported by Maruyama deals with the dental examination of 1511 native Chinese children ranging from 1 to 15 years of age. These examinations were conducted in Taihoku, which is the capital of Formosa (Taiwan) and the seat of a medical school established by the Nipponese. Interpreters were employed by the examiners to secure information from the Formosans, who speak a local Chinese dialect. The inspection of the teeth was performed with the aid

[1] Received for publication March 26, 1946.

of dental mirrors and probes. The examiners were apparently members of the dental department of the Garrison Hospital in Taihoku.

The records obtained were divided into 4 groups for analysis of the material: 1) incompletely erupted deciduous dentitions of 129 boys and 104 girls 1 to 3 years of age, inclusive; 2) fully erupted deciduous dentitions of 296 boys and 280 girls 3 to 8 years of age; 3) mixed dentitions of 234 boys and 221 girls 6 to 10 years of age; and 4) mixed dentitions of 146 boys and 101 girls 11 to 15 years of age. The entire group thus consisted of 805 boys and 706 girls.

The most interesting statistical information contained in the 35 tables in the original report can be conveniently summarized by the construction of graphs.

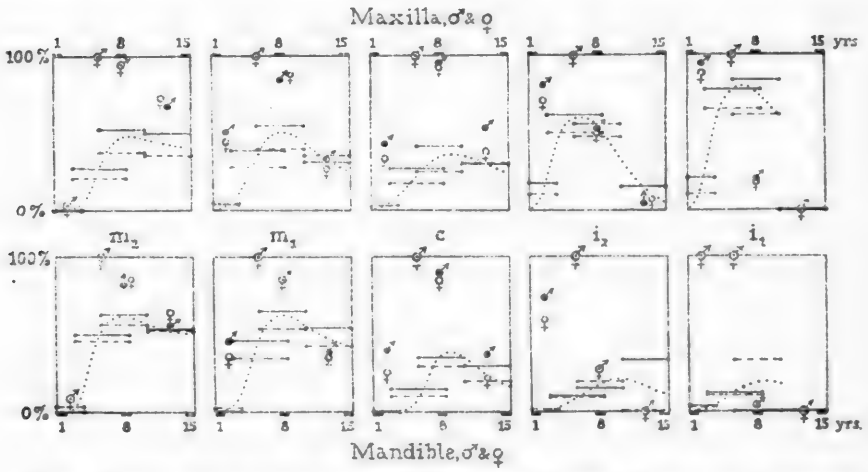

Fig. 1. Graphed data on deciduous teeth of Formosa Chinese. *Sex symbols:* percentages of erupted teeth present in each age group, based on maximum number possible if each individual eventually erupts a normal number of teeth. *Horizontal lines:* percentages of unexfoliated erupted teeth found to be necrotic. *Dotted line curves:* smoothed trends suggested by averaging values indicated by horizontal lines. (Example. About 37% of the boys and 22% of the girls in the 11 to 15 year old age group had unexfoliated mandibular canines. Almost 30% of these teeth were decayed in the males and 20% in the females.)

Fig. 1 presents the percentages of decayed deciduous teeth in the 4 clinical groups. These data were obtained from Tables 4, 6, 14 and 17 in Maruyama's report. The solid horizontal lines indicate the percentages of unexfoliated erupted teeth that were found to be decayed in boys, while the corresponding percentages for girls are shown by the broken lines. Where the values for the sexes coincide, the broken lines are omitted. The percentages of erupted teeth of a particular morphological variety present in each clinical group were computed from data in Tables 1, 4, 6, 7, 10, 14 and 17 in the original report. These percentages may be looked upon as being approximately representative of the percentages of children in each clinical group with (unexfoliated) erupted teeth of a particular anatomical classification.

The dotted curves indicate a summarization of the general trends suggested by studying the mean percentage values computed from the data on the decayed teeth of each sex in all 4 clinical groups. With one possible exception (the mandibular first incisor) all of these curves ascend to a maximum somewhere between the sixth and ninth years of life, after which they descend toward the base line. The steepness of the descent is never as marked as that of the ascent.

It is difficult to verify these findings by further study of Maruyama's report, as he tabulates almost none of his figures on the basis of 1 year age periods. Table 9 in his paper, however, contains a more detailed analysis of the second clinical group with fully erupted deciduous dentitions, although the data relating to the maxillary teeth are combined with those relating to the corresponding mandibular teeth. *Fig. 2* gives these data graphically, the dots representing the percentages of decayed teeth in the males and the circles the percentages in the females. The average values for the sexes are represented by the solid-line

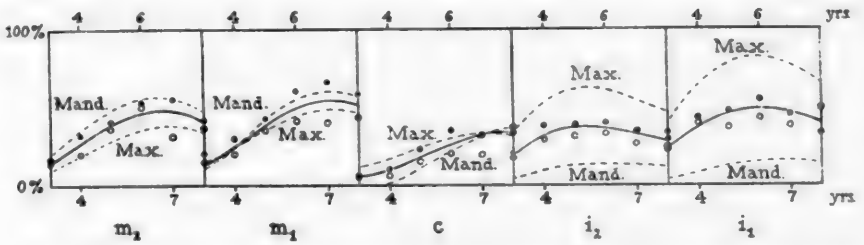

Fig. 2. Graphed data on the deciduous teeth of Formosa Chinese with complete deciduous dentitions. *Dots:* percentages of decayed teeth in males. *Circles:* same, in females. *Solid lines:* trends suggested by average values for both sexes. *Broken lines:* division of values represented by solid lines into maxillary and mandibular components.

curves, which were drawn free-hand in such a manner as to smooth out minor irregularities. The broken-line curves on each side of the solid lines and equidistant from them at any given point, if the measurements are made along the vertical coördinates only, represent an estimated division of the average values into maxillary and mandibular components. These estimates were based upon the relative frequencies of decay shown in *fig. 1*.

This detailed analysis of the data on 576 children with complete deciduous dentitions confirms the probable correctness of the summarization curves shown in *fig. 1*. The majority of the individual frequencies depicted in *fig. 2* reach a maximum value somewhere between the sixth and seventh years of life; in the case of the canines the maxima seem to occur a year or 2 later.

These analytical findings are of considerable interest in view of the fact that almost none of the statistical tabulations on deciduous teeth available today give one any reason to suspect that sizeable groups of children past a certain critical age period and selected at random[2] may have smaller percentages of decayed teeth than some of the younger age groups, especially if the statistics deal with affected

[2] Except for number of unexfoliated teeth.

individual teeth instead of affected dentitions. The macroscopic dental signs of disease are not known to disappear completely even in those cases in which the teeth exhibit an arrest of pathologic processes. It is hardly necessary to point out in this connection that the few reports which give information on the percentages of children at various ages with decayed, filled, or surgically removed deciduous teeth are of no value in studies of this kind. Not only does the failure to present the data on the basis of the different classes of teeth involved detract from the usefulness of these reports, but their worth is minimized further by the absence of information on the natural exfoliation of the teeth. The paper by Stoughton and Meaker (17) can be cited as an example of this type of statistical report.

The report by Ottofy (12) on 500 Filipino children examined in 1902 contains data that furnish particularly valuable clues for interpreting the figures published by Maruyama. In addition to tabulating the number of sound and diseased deciduous teeth seen in each group of children, Ottofy provides similar data on the permanent teeth seen in the same groups of individuals. It is therefore possible to relate the total number of unexfoliated deciduous teeth of a given anatomical classification to the total number of their erupted permanent successors and to compute the percentages of decayed deciduous teeth both on the basis of the number of unexfoliated teeth and the number of erupted (deciduous) teeth in the same age groups.

Fig. 3 presents a simplified graphic summary of these data for 3 classes of deciduous maxillary teeth. The upper section of the figure gives the percentages of decayed[3] (d) and sound (s) teeth calculated in the same manner as Maruyama's percentages, that is, on the basis of the number of unexfoliated erupted teeth. The general trends suggested by the yearly percentages are indicated by the solid lines, which were drawn free-hand to provide the lines of best fit to the 3 series of individual values. The lower section of the figure summarizes the percentages of sound and decayed deciduous teeth calculated on the basis of the total number of dentitions, it being assumed that each dentition possessed a normal number of all classes of teeth. These percentages correspond roughly to the percentages of children in each age group with sound and diseased unexfoliated deciduous teeth. The percentages of sound and decayed (SD) permanent teeth are indicated by the dotted-line curves, which were drawn free-hand to show the curves suggested by the crosses (X). Except in the case of the second premolars, these curves are suggestive of a 3 to 6 month average interval between the emergence of the permanent teeth and the exfoliation of their deciduous predecessors. These intervals correspond roughly with those that can be determined from the data on Scottish children published by King (10) in 1940. The percentages of deciduous teeth present in the Filipino children are shown by the dotted-line curves which are suggested by the plus signs (+).

The curves shown in the upper sections of *fig. 3* are strikingly similar to the summarization curves obtained from Maruyama's material, although exhibiting less pronounced descents than the latter. With the exception of the mandibular

[3] Extracted and filled teeth were counted as decayed teeth by Ottofy.

incisor teeth, similar curves can be obtained for all other deciduous teeth from the data reported by Ottofy. (The mandibular incisor curves are nearly straight horizontal lines near the base line.) After the ninth year of life, or earlier, the percentages of sound unexfoliated deciduous teeth increase, while there is a corresponding diminution in the percentages of decayed deciduous teeth. By combining the study of these percentages with an analysis of the data pertaining to exfoliation and eruption, as shown in the lower sections of *fig. 3*, it appears that there may be some association between the disturbances responsible for dental decay and the growth processes responsible for the exfoliation of the deciduous

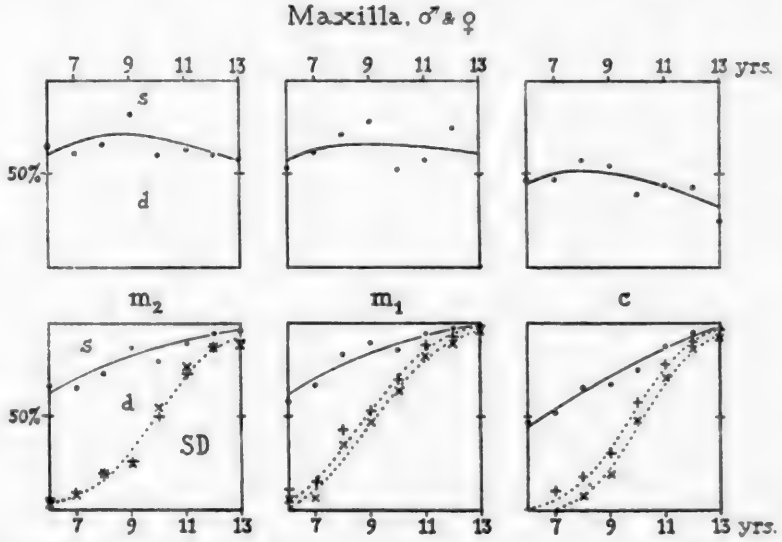

Fig. 3. Graphed data on the deciduous maxillary molars and canines of Filipinos. Upper section (*solid lines*): percentages of sound (s) and necrotic (d) unexfoliated erupted teeth, in terms of absolute values found and general trends suggested. Lower section: a. *solid lines:* percentages of sound and necrotic teeth, calculated on the basis of the maximum number possible if every individual eventually erupts a normal number of teeth; b. *dotted lines:* exfoliation of deciduous teeth (+'s) and eruption of their permanent successors (x's).

teeth. Necrotic deciduous teeth (or dentitions) seem to be eliminated more quickly than sound teeth (or dentitions). Thus there is a possibility that among the natives of Philippine Islands rapid physical maturation may favor the development of lesions in the calcified tissues of the teeth.

The data by Maruyama, shown graphically in *fig.* 2, do not necessitate a different explanation. Since these data are based on an examination of children with complete deciduous dentitions, it stands to reason that while this clinical group includes children who were slow to exfoliate their deciduous teeth it also excludes those who had lost some of these teeth early. There is considerable over-lap of groups II and III due to the fact that a few 6-year old children have already lost

some of their deciduous teeth while a few 8-year olds still possess complete deciduous dentitions. Table I, which was prepared from data in Tables 1, 7 and 10 in Maruyama's report, illustrates this point quite clearly. If it is true that the late eruptors as a group have better teeth than the early eruptors, then it is to be expected that the summarization curves shown in *fig. 2* should fall whenever a sufficiently large proportion of the data on the early eruptors' teeth has been segregated from the total data. This does not occur until during the 7th and 8th years of life in most classes of deciduous teeth.

Partial confirmation of these findings on Far Eastern clinical material can be obtained by examining the data by Cohen (7) on a racially heterogenous group of Minneapolis children. Lack of space prohibits an adequte analysis of this North

TABLE I

The number of children examined

AGE	GROUP I	GROUP II	GROUPS III AND IV	TOTALS
years				
1	60			60
2	135			135
3	38	77		115
4		152		152
5		147		147
6		138	6	144
7		52	69	121
8		10	118	128
9			132	132
10			130	130
11			116	116
12			65	65
13			45	45
14			15	15
15			6	6
Totals.....	233	576	702	1511

American material here. The tentative explanation offered in this report cannot be invalidated by the argument that those who lose their deciduous teeth early may erupt them early and therefore have more decay at all age levels than those whose dentitions develop at a slower rate. If the amount of necrotic odontoclasia seen in the clinic depended primarily on the period of mouth exposure of the teeth, then those who erupt their teeth later than average should have as much decay as others, provided that proper allowance is made for the time factor in such comparisons. In no case should it be possible to obtain summarization curves of the type discussed in this report. Moreover, it should be borne in mind that the exposure risks of the deciduous teeth of late eruptors are always likely to be greater than those of early eruptors. The variation in the eruption times of deciduous teeth is generally very much less than in the case of permanent teeth. A simple method of convincing one's self of the actuality of this fact is by comparing

the standard deviations of the age distributions of eruption of the 2 dentitions. Table II was prepared from data from 3 sources: (a) those by Palmer, Klein and Kramer (13) on the permanent teeth of 4,416 white children examined in Hagerstown, Maryland; (b) those by Boas (6) on the deciduous teeth of 1,167 Hebrew infants examined in New York City; and (c) those by Robinow, Richards and Anderson (14) on 1,980 white children observed at the Fels Research Institute in Yellow Springs, Ohio. Inasmuch as great accuracy is not essential for the purposes of this comparison, the values for boys were averaged with those for girls. The standard deviations obtained from Boas' data are based on both computations and estimates, as the original figures are given in terms of months and calculated according to 2 different procedures. Since the standard deviations

TABLE II

Averaged standard deviations of the ages of eruption of the permanent and deciduous teeth of Caucasian males and females in the United States

PERMANENT TEETH	STAND. DEV. ACCORDING TO PALMER ET AL.	DECIDUOUS TEETH	STAND. DEV. ACCORDING TO		RATIOS BASED ON DATA BY	
			Boas	Robinow et al.	Boas	Robinow et al.
	years		*years*	*years*		
Max. I_1	0.75	Max. i_1	0.25	0.15	3.0:1	5.0:1
Max. I_2	1.02	Max. i_2	0.29	0.21	3.5:1	4.9:1
Max. C	1.40	Max. c	0.40	0.25	3.5:1	5.6:1
Max. Pm_1	1.45	Max. m_1	0.28	0.19	5.1:1	7.6:1
Max. Pm_2	1.64	Max. m_2	0.41	0.37	4.0:1	4.4:1
Mand. I_1	0.72	Mand. i_1	0.24	0.15	3.0:1	4.8:1
Mand. I_2	0.78	Mand. i_2	0.35	0.42	2.2:1	1.9:1
Mand. C	1.15	Mand. c	0.39	0.26	3.0:1	4.4:1
Mand. Pm_1	0.81	Mand. m_1	0.30	0.17	2.7:1	4.8:1
Mand. Pm_2	1.38	Mand. m_2	0.41	0.33	3.4:1	4.2:1
Average.....	1.11		0.33	0.25	3.34:1	4.76:1

given by Boas and Robinow, Richards and Anderson show considerable discrepancies, the ratios obtained by comparing the values for the deciduous teeth with those for the permanent ones were calculated separately for these 2 series of data.

As shown by the ratios in Table II, the age distributions of eruption of the permanent teeth are on an average from 3 to 5 times as extensive as those of the deciduous teeth. It is, of course, to be expected that absolute variability as measured by the size of the standard deviations would increase with age. Thus it is obvious that late exfoliation of the deciduous teeth is almost never preceded by equally late eruption.

A study of Ottofy's data leads one to the conclusion that the permanent teeth of Filipinos emerge on an average $\frac{1}{2}$ to $1\frac{1}{2}$ years earlier than those of Caucasians living in temperate zones. There is no reason for supposing that the eruption times of Formosa Chinese are markedly different from those of Filipinos who live

in a similar climate some 700 miles south. The report by Suk (18) indicates an appreciable difference between the eruption times of Zulus living in Africa and those of Bohemians living in Europe. The findings of Spier (15) and of Steggerda and Hill (16) likewise strengthen the impression that the populations native to hot climates generally erupt their permanent teeth earlier than those native to cooler regions. Nevertheless, the more one studies the meager statistical data now available on dental development and disease, the more one hesitates to draw the conclusion that rapidly maturing ethnic groups are particularly susceptible to dental deterioration. Such a conclusion is unwarranted today in view of our limited knowledge regarding the genetic characteristics of various strains of *Homo sapiens*, not to mention certain other difficulties pointed out by Boas (5).

One may be tempted to say that it is within the realm of possibility that disease involving the deciduous teeth may act as a stimulant for the eruption of the permanent teeth. A hypothesis of this kind ignores the fundamental philosophy of the causation of disease. In the words of Draper (8) "disease arises from the interplay of dynamic forces inherent in the individual and present in the world about him", which means that all pathological reactions are evidence of some disequilibrium between the vital forces of an organism and certain environmental conditions. The vital forces of a slowly growing organism or organ are not the precise equivalent of those of a rapidly growing organism or organ at any given moment of time. Therefore, it is inconceivable that rapidly growing bodies could react to all environmental stimuli in precisely the same manner as the more slowly growing ones. The environmental conditions which are barely able to cause pathological changes in one group of organisms cannot be expected to be able to initiate like changes in an unlike group of organisms.

It would be nonsensical to try to maintain that any disease, including dental caries, could act as an independent variable that can modify the same vital forces which under certain environmental conditions made the development of the disease inevitable. The vitality of a tooth, or the growth force of a tooth bud, cannot be altered by any disease whatsoever for the simple reason that "disease" is an abstraction, while the tooth is a concrete fact. "Disease" is a verbal invention for expressing the condition of faulty living tissues.

Some of the conclusions reached by Robinow, Richards and Anderson (14) are of special interest in concluding this interpretative analysis of Far Eastern data. These authors found, as a result of statistical studies involving an inspection of factor loadings and communalities, that the eruption of deciduous teeth had a low correlation with certain signs of maturation, such as the appearance of ossification centers, height and onset of walking. They state, furthermore, that "eruption depends comparatively little on conditions unique for any individual tooth pair . . . The high communalities suggest that environmental factors are not as important as might be suspected." Necrotic lesions in enamel and dentin certainly constitute conditions which are unique for any individual tooth pair. Therefore they should not, by themselves, have any appreciable influence on the processes of eruption.

SUMMARY

1. Virtually complete lack of medical and dental services for children in the Far East makes statistical surveys of health conditions in this region particularly informative.

2. Rapid growth of the masticatory organs of children may favor the development of necrosis of the enamel and dentin of deciduous teeth. Conversely, slow dental maturation tends to be associated with freedom from caries in early childhood.

3. It is not possible to explain the higher frequencies of decay of the deciduous teeth of early eruptors on the basis of longer periods of mouth exposure.

4. The apparent relationship between growth and dental disease is readily demonstrable only in racially homogeneous groups. The genetics of man is still too poorly known to permit free comparison of dissimilar or markedly heterogeneous ethnic groups.

5. It would be illogical to maintain that decay of the deciduous teeth may accelerate the eruption of their permanent successors.

The author wishes to express his appreciation to Mr. H. Minatoya of the Department of Physiology at the College of Medicine, University of Illinois, for his invaluable assistance in translating portions of the article by Maruyama.

REFERENCES TO LITERATURE

1. Agnew, M. C., Dietary studies in Szechwan. *J. West China Border Research Society*, **3**: 55, 1926–1929.
2. Agnew, R. G., Oral pathology in Szechwan. *J. West China Border Research Society*, **3**: 61, 1926–1929.
3. Anderson, B. G., A study of the incidence of dental caries in one thousand Chinese. *J. D. Res.*, **12**: 709, 1932.
4. Ballantine, J. W., I lived on Formosa. *National Geographic Magazine*, **37**: 1, 1945.
5. Boas, F., Some notes on the eruption of teeth. *D. Cosmos*, **62**: 390, 1920.
6. *Idem*, The eruption of deciduous teeth among Hebrew infants. *J. D. Res.*, **7**: 245, 1927.
7. Cohen, J. T., A statistical study of caries in the deciduous and permanent teeth of children. *J. A. D. A.*, **23**: 312, 1936.
8. Draper, G., *Human Constitution*. (Philadelphia and London, W. B. Saunders Co., 1924.)
9. Maruyama, Y., Report I: Statistical studies of decay of the deciduous teeth and the author's objections to hitherto employed methods of approaching the problem. *Taiwan Igakkai Zasshi* (Journal of the Medical Association of Formosa), **31**: 1289, 1932.
10. Medical Research Council (Great Britain): King, J. D., Dental Disease in the Island of Lewis. *Special Report No. 241* (London, His Majesty's Stationery Office: 1940).
11. Montelius, G. A., Observations on teeth of Chinese. *J. D. Res.*, **13**: 501, 1933.
12. Ottofy, L., International examination and tabulation of the condition of the teeth of public school children. *Transactions of the Fourth International Dental Congress*, **2**: 71, 1905.
13. Palmer, C. E., Klein, H., and Kramer, M., Studies on dental caries. III. A method of determining post-eruptive tooth age. *Growth*, **2**: 149, 1938.
14. Robinow, M., Richards, T. W., and Anderson, M., The eruption of deciduous teeth. *Growth*, **6**: 127, 1942.

15. Spier, L., The growth of Porto Rican boys, with special reference to the relation between their stature and dentition. *J. D. Res.*, **1**: 145, 1919.
16. Steggerda, M., and Hill, J. T., Eruption time of teeth among whites, Negroes and Indians. *Am. J. Orthod. and Oral Surg.*, **28** (Orthod.): 361, 1942.
17. Stoughton, A. L., and Meaker, V. T., Dental decay and corrections among school children of different ages. *Public Health Rep.*, **46**: 2608, 1931.
18. Suk, V., Eruption and decay of permanent teeth in whites and Negroes, with comparative remarks on other races. *Am. J. Phys. Anthrop.*, **2**: 351, 1919.

HYDROLYZED SALIVA AS A SOURCE OF NUTRIENTS ESSENTIAL FOR THE PRODUCTION OF ACID BY AN ORAL STRAIN OF LACTOBACILLUS ACIDOPHILUS[1]

DAVID WEISBERGER, D.M.D., M.D.

Harvard School of Dental Medicine, Boston, Mass.

It has been shown (1) that an oral strain of *Lactobacillus acidophilus* will produce acid to a maximal extent when grown on the synthetic medium described by Landy and Dickens (2). At the same time it was found that not all of the constituents listed by these authors are essential for this process. The organism produced acid to a maximal extent when grown upon a simplified medium consisting of casein hydrolyzate, tryptophane, calcium pantothenate, nicotinic acid, thiamine hydrochloride, glucose, potassium mono- and di-phosphates[2], and sodium acetate.

Later (3) it was shown that paraffin stimulated whole saliva could be used in place of the inorganic phosphate of this synthetic medium. Also it was found that saliva, if concentrated, could be substituted for the calcium pantothenate, nicotinic acid, or the thiamine, but the acid production by *Lactobacillus acidophilus* was at less than the maximal rate. On the other hand when saliva, concentrated as much as 7 fold, replaced the casein hydrolyzate or the tryptophane no acid production resulted. As a consequence of these findings it was concluded (3) that the mineral salts and the vitamins essential for at least some acid production were present in normal saliva. However, the nitrogenous substances necessary for acid production either were not present in sufficient quantities in the concentrated saliva or were not in an available form.

Obviously it was of interest to learn whether hydrolyzed saliva would require fewer or more supplements than had concentrated whole saliva when it assisted *Lactobacillus acidophilus* in producing acid. Therefore saliva which had been hydrolyzed by acid was used as a replacement in turn for each of the ingredients of the medium of Landy and Dickens (2) and the acid produced by the organism in each instance was determined. Later saliva hydrolyzed by alkali was tested as a replacement for the nitrogenous constituents of a synthetic medium simpler than that described by Landy and Dickens. The observations made in the course of these experiments are outlined in this report.

EXPERIMENTAL

In general the methods and materials used in isolating and cultivating the organism and in preparing stock solutions and test media were similar to those described in the first paper (1) of this series. The preparation of the test

[1] Received for publication March 14, 1946.

[2] Hereafter designated as Salt mixture A.

experiments, and the adjustment of pH, the autoclaving, inoculation, incubation of the individual cultures and the procedure used in the titration of the acid formed have all been described in the same place.

(*A*) *The Action of Acid Hydrolyzate of Saliva*

Preparation of the Hydrolyzate of Saliva. 500 ml. of saliva was collected from 5 subjects chosen from laboratory workers. Paraffin chewing was used to aid secretion of the fluid. The whole saliva was filtered through glass wool to remove the grossly particulate matter. Then it was hydrolyzed in a manner substantially the same as that outlined by Mueller and Johnson (4). The filtered saliva was mixed with an equal volume of concentrated hydrochloric acid and transferred to a 1 liter round bottom flask. This was connected to a water cooled reflux condenser and the mixture was heated on an electric hot plate. It was maintained at the boiling temperature for 18 hours. Then the acid mixture was placed in a vacuum distillation apparatus. The solution was evaporated just to dryness with the aid of an efficient vacuum pump and a water bath maintained at a temperature just sufficient to cause ebullition in the evaporating liquid. Then 200 ml. of water was added which dissolved most of the concentrate and the solution was again evaporated to dryness in vacuo. After this second evaporation 100 ml. of distilled water was added, the mixture was agitated for several minutes and then filtered through a fine grade of filter paper. The filtrate was made up to a volume of 100 ml. and its nitrogen content was determined. Per ml. it contained 3.4 mg. total nitrogen (Kjeldahl method (5)), 3.15 mg. nitrogen not precipitable by phosphotungstic acid, and 1.5 mg. amino acid nitrogen (Van Slyke method (6)).

Acid Hydrolyzate of Saliva as a Source of Nutrients for Acid Production by Lactobacillus acidophilus. A medium containing in double strength all ingredients listed by Landy and Dickens except casein hydrolyzate was prepared. To 5 ml. of this we added in one instance 0.25 ml., in another 0.5 ml. of the acid hydrolyzate of saliva. In a third instance 0.25 ml. of casein hydrolyzate (Smacovitamin free) was added. Then the test mixture was diluted to 10 ml. with distilled water, was autoclaved, inoculated, incubated and the acid produced by the organism was titrated. The quantity of casein hydrolyzate added corresponded to that present in 10 ml. of Landy and Dickens culture medium. The measurements of the acid produced are presented in Table I.

In additional experiments the capacity of acid hydrolyzed saliva to replace the ingredients other than the casein hydrolyzate of Landy and Dickens medium was tested. For these, double strength solutions lacking casein hydrolyzate and, in turn, 1 other of the constituents listed in Table II were employed. In the experiments 5 ml. portions of each stock solution were measured into culture tubes, 0.5 ml. of acid hydrolyzate of saliva was added, the mixtures diluted to 10 ml. and the test carried out as described above. At the same time comparable tests were made in which the ingredient being investigated was present. The results of experiments of this series are shown in Table II. Finally an experiment was performed in which in addition to acid hydrolyzate of saliva the substances listed

TABLE I

*Comparison of acid produced when acid hydrolyzate of saliva is substituted for casein hydrolyzate**

CASEIN HYDROLYZATE ADDED	ACID HYDROLYZATE OF SALIVA ADDED	AMOUNT OF ACID (0.1 N) PRODUCED
ml.	*ml.*	*ml.*
none	0.25	2.30
none	0.50	4.78
0.25	none	5.00

Basal medium contained all ingredients listed by Landy and Dickens (2) except casein hydrolyzate.

* Total volume of culture media used was 10 ml.

TABLE II

*Acid produced when nutrients of the medium of Landy and Dickens (2) were tested as supplements for an acid hydrolyzate of saliva**

NUTRIENTS INVESTIGATED†	NUTRIENT ABSENT FROM MEDIA	NUTRIENT PRESENT IN MEDIA
	ml. of 0.1 N acid	*ml. of 0.1 N acid*
Essential		
Tryptophane	0.00	4.70
Calcium pantothenate	0.31	4.58
Nicotinic acid	1.20	4.62
Thiamin hydrochloride	0.17	4.71
Glucose	0.00	4.57
Non-essential		
Casein hydrolyzate	4.78	4.79
Asparagine	4.77	4.70
Cystine	4.67	4.71
Guanine hydrochloride	4.40	4.49
Adenine sulfate	4.46	4.51
Xanthine	4.50	4.63
Uracil	4.40	4.76
Biotin	4.62	4.67
Riboflavin	4.54	4.81
Folic acid	4.71	4.72
Pyridoxine hydrochloride	4.62	4.48
Salt solution A	4.82	4.85
Salt solution B	4.81	4.84
Essential Substances only		4.75
Complete media		4.78

* Total volume of each culture was 10 ml.
0.5 ml. of acid hydrolyzate of saliva was present in each.
† Sodium acetate was present in each culture.

in Table II as essential plus casein hydrolyzate were all present. This was compared with another test which contained all ingredients, except the casein hydrolyzate, of the complete Landy and Dickens medium. The quantity of acid produced was substantially the same in both instances (see Table II).

(*B*) *The Action of Alkaline Hydrolyzate of Saliva*

Preparation of Alkaline Hydrolyzate of Saliva. 500 ml. of saliva was collected from subjects chewing paraffin. To this we added 500 ml. of 3.7 N hot solution of barium hydroxide. The mixture was placed in a flask, connected to a reflux condenser and boiled on an electric hot plate for 10 hours. The solution was cooled, placed in an icebox overnight and then filtered to remove the precipitated barium hydroxide. The precipitate was washed with four small portions of ice

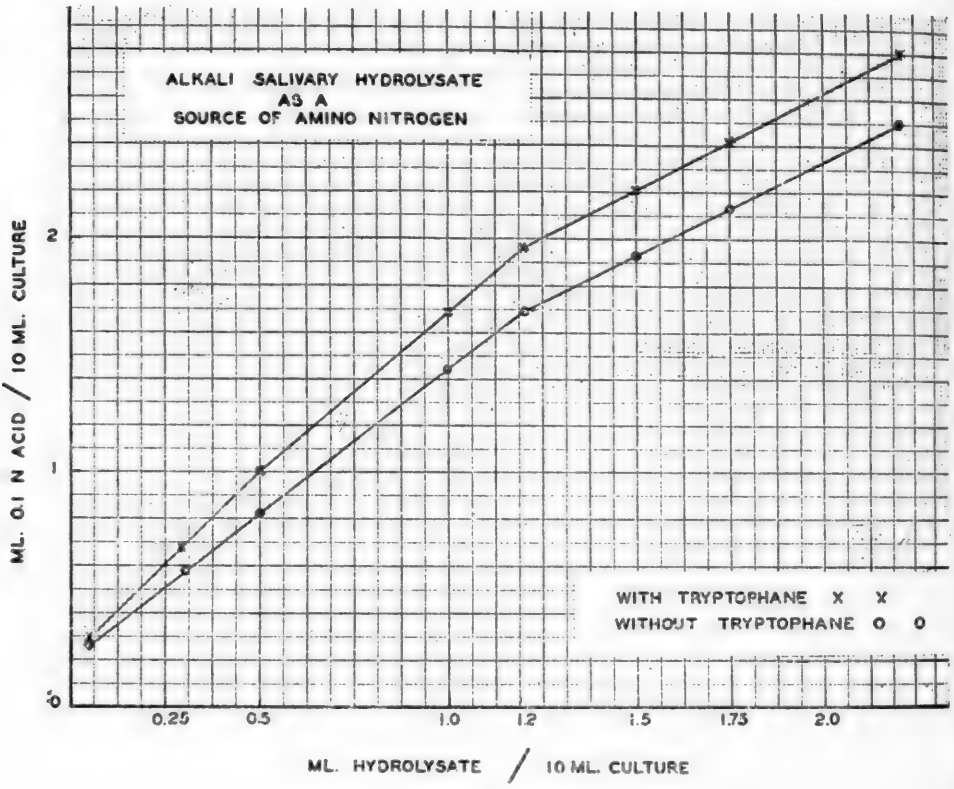

Fig. 1

cold water. The filtrate was concentrated in vacuo just to dryness. The residue was taken up in 100 ml. of water, agitated gently for a few minutes, and then filtered through a fine grade of filter paper. The amino acid nitrogen content of this alkaline hydrolyzate was 1.28 mg. per ml. and the total nitrogen was 2.52 mg. per ml.

Alkaline Hydrolyzate of Saliva as a Source of Nutrients for Acid Production by Lactobacillus acidophilus. For experiments of this series a stock solution containing glucose, sodium acetate, mono- and di-potassium phosphates, thiamine hydrochloride, calcium pantothenate and nicotinic acid was prepared.

The ingredients were present in double their concentrations in the medium of Landy and Dickens. From this solution a series of cultures were prepared. Each of these contained 5 ml. of the stock and, in addition, quantities of alkaline hydrolyzate of saliva varying from 0.04 to 2.2 ml. Then a second series of cultures were made up in a similar manner with the addition of 1.0 mg. of l-tryptophane to each tube. A third set of cultures was made up containing

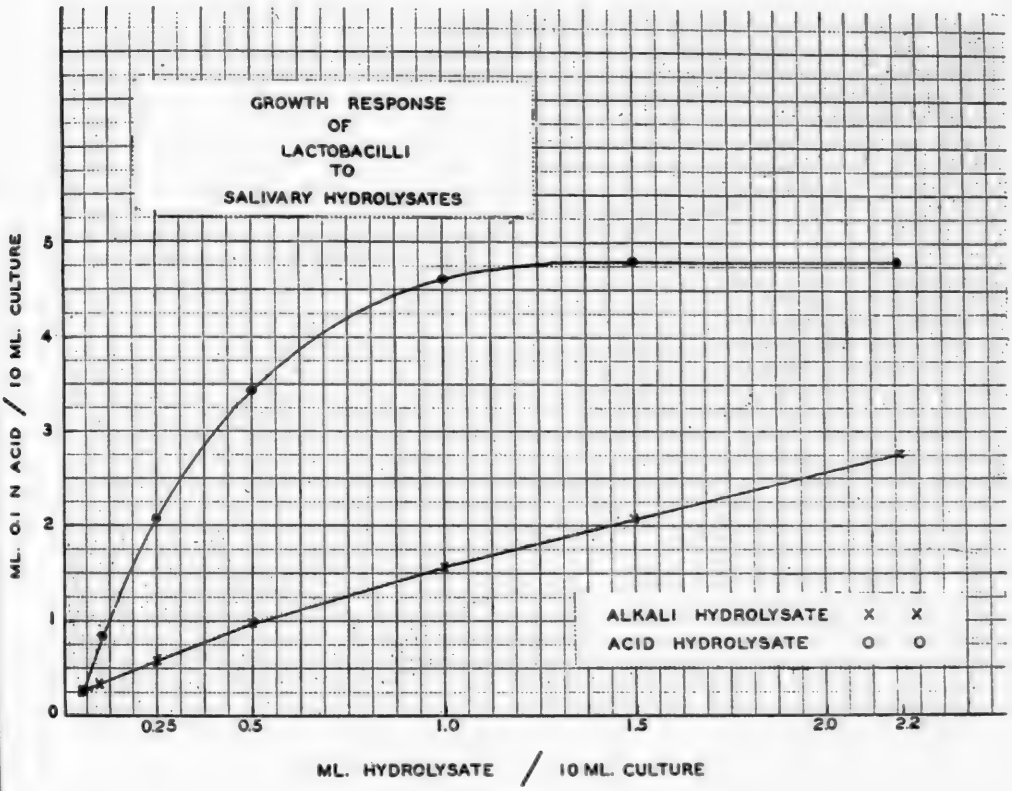

Fig. 2

casein hydrolyzate and the substances listed in Table II as essential for maximal acid production by this organism. This series was used as a control on the activity of the organism. The cultures of all series were treated to adjust pH to 6.8, made up to 10 ml. volume, autoclaved, inoculated, incubated and titrated in the manner described previously. The measurments of the acid produced in media containing varying amounts of an alkaline hydrolyzate of saliva as the source of the nitrogenous substances are presented in *Fig. 1*. The slight additional effect of added tryptophane is shown in the same place.

Since it was shown during this investigation that acid hydrolyzate of saliva could function as a substitute for casein hydrolyzate of simplified synthetic

media and that alkaline hydrolyzate of saliva could replace both casein hydrolyzate and tryptophane in such media, it was of interest to compare the relative effectiveness of these 2 hydrolyzates in promoting acid production by *Lactobacillus acidophilus*. A number of tubes containing 5 ml. portions of a double strength stock solution containing all ingredients previously found essential except casein hydrolyzate were set up. To one group of these, portions of acid hydrolyzate of saliva in quantities from 0.04 to 2.2 ml. were added. To another group similar quantities of alkaline hydrolyzate of saliva were added. All tubes were treated to bring the pH to 6.8, volumes were adjusted to 10 ml., autoclaved, inoculated with the organism, incubated for 72 hours and the acid produced was titrated. The results are shown in *Fig. 2*.

RESULTS

The data in Table I show that an acid hydrolyzate of saliva can function as a replacement for casein hydrolyzate in preparing a medium in which *Lactobacillus acidophilus* can produce acid. With 0.25 ml. of the saliva hydrolyzate, which contributed 0.75 mg. of amino acid nitrogen and 1.70 mg. of total nitrogen, the acid produced was approximately half the quantity obtained in the control experiment with the 0.25 ml. of casein hydrolyzate (Smaco) which provided 2.95 mg. of amino acid nitrogen and 2.5 mg. of total nitrogen. When the quantity of saliva hydrolyzate was doubled the amount of acid produced approximated that obtained with the casein hydrolyzate. Thus it appears that per unit of amino acid nitrogen saliva hydrolyzate is more potent than casein hydrolyzate in promoting the production of acid by this organism.

The data in Table II demonstrate that acid hydrolyzed saliva did not function as a replacement for l-tryptophane or for glucose in promoting maximal acid production by *Lactobacillus acidophilus*. It did, however, have capacity to aid in the production of small quantities of acid when the vitamins, thiamine hydrochloride, pantothenic acid and nicotinic acid were omitted. The quantities of acid produced by the organism when these vitamins were absent and when acid hydrolyzed saliva was present were approximately the same as those in our previous investigation (3) where whole concentrated saliva occupied the position of the acid hydrolyzed saliva in this study. Together these observations support the conclusion that concentrates of whole and of hydrolyzed saliva contain small amounts of these vitamins.

The data in Table II also show the supplements found to be necessary for maximal acid production by *Lactobacillus acidophilus* when acid hydrolyzed saliva is substituted for casein hydrolyzate. These are the same as those found to be essential in the previous investigation (1) when a simplified synthetic medium upon which *Lactobacillus acidophilus* would grow was described.

The data obtained with alkaline hydrolyzate of saliva are portrayed in *Fig. 1*. There it can be seen that an alkaline hydrolyzate of human saliva by itself can be used as a replacement for both the casein hydrolyzate and the tryptophane of the synthetic medium in promoting the production of titratable acid. Addition of 1.0 mg. of tryptophane to the medium containing the alkaline hydrolyzate of

saliva increased the acid production only slightly at all concentrations of the hydrolyzate. The concentration of the hydrolyzate rather than the tryptophane seemed to be the limiting factor in these experiments. Together these findings indicate the presence of free tryptophane in alkaline hydrolyzate of saliva and thus support our previously recorded observation (3) regarding the positive color reaction for combined tryptophane given by whole saliva. The failure of acid hydrolyzed saliva to act as a substitute for tryptophane in the media used in the experiments of Table II can be understood on the basis of well established observations that acid hydrolysis of protein is accompanied by the destruction (7) of any tryptophane present in the original protein. These findings also indicate that free tryptophane must be present when *Lactobacillus acidophilus* is producing acid.

Acid and alkaline hydrolyzates of saliva having similar amino acid nitrogen contents, approximately 1.25 mg. per ml., were compared with respect to their effectiveness in promoting the production of acid by *Lactobacillus acidophilus*. It was found (*Fig. 2*) that the acid hydrolyzate was more efficient per unit of volume and reached its maximum with the presence of 1.0 ml. of hydrolyzate. The effect of the alkaline hydrolyzate increased linearly as the amount of the hydrolyzate present was enlarged. The maximum effect had not been achieved when 2.2 ml. of the alkaline hydrolyzate was present. Since the amino acid nitrogen content of the 2 hydrolyzates was similar and since the other ingredients of the media were the same it is necessary to seek some explanation for the differences in the quantity of acid produced with the aid of these hydrolyzates. One possibility is that the racemization of amino acids, which is known to occur during hydrolysis by alkali (8) has reduced the concentration of the active form of the amino acids sufficiently to slow up production of acidity by the organisms. Another possibility is that the alkaline hydrolysis has destroyed a portion of some amino acid, such as cystine (8) which may be necessary for the activity of the organism. A third possibility is that barium ions, which our preparative procedure did not completely remove had an inhibiting effect. This possibility was eliminated by a control experiment in which excess barium was removed by dilute sulphuric acid. The saliva hydrolyzate from which excess barium was not removed yielded 5.23 ml. of 0.1 N acid while that from which the barium was removed produced 5.47 ml. of acid.

SUMMARY

1. Saliva hydrolyzed by acid or by alkali has been successfully substituted for casein hydrolyzate as a nutrient in several simplified synthetic media which promote acid production by *Lactobacillus acidophilus*. This is in contrast to the earlier observation that whole saliva could not be substituted for casein hydrolyzate.

2. It was not necessary to supply l-tryptophane to the synthetic media when an alkaline hydrolyzate of saliva was present. This finding is in contrast to the earlier observation (3) that whole saliva could not be substituted for free tryptophane. Thus the presence of free tryptophane in alkaline hydrolyzate of saliva

is indicated and the previous observation regarding its presence in a combined form in whole saliva is given additional support.

REFERENCES TO LITERATURE

1. Weisberger, D., and Johnson, F. G., Nutritional requirements for an oral strain of Lactobacillus acidophilus. *J. D. Res.*, **25**: 35, 1946.
2. Landy, M., and Dickens, D. M., Microbiological assay method for six B. vitamins using Lactobacillus casei and medium of essentially known composition. *J. Lab. and Clinical Med.*, **27**: 1086 (1942).
3. Weisberger, D., Whole saliva as a source of certain growth factors for an oral strain of Lactobacillus. *J. D. Res.*, **25**: 85 (1946).
4. Mueller, J. H., and Johnson, E. R., Acid hydrolyzates of casein to replace peptone in the preparation of bacteriological media. *J. Immunol.*, **40**: 33 (1941).
5. Folin, O., and Wright, L. E., A simplified macro-Kjeldahl method for urine. *J. Biol. Chem.*, **38**: 461 (1919).
6. Peters, J. P., and Van Slyke, D. D., Quantitative Clinical Chemistry, Methods. (Baltimore, Williams & Wilkins Co.: 1932) p. 386.
7. Schmidt, C. L. A., The Chemistry of the Amino Acids and Proteins, 2nd ed., (Springfield, Ill., Charles C. Thomas Co.: 1944) pp. 126, 129, 130.
8. *Ibid.*, pp. 58 and 130.

THE APPOSITIONAL LIFE SPAN OF THE ENAMEL AND DENTIN-FORMING CELLS

I. Human Deciduous Teeth and First Permanent Molars[1]

INTRODUCTION

MAURY MASSLER, D.D.S., M.S., and ISAAC SCHOUR, D.D.S., Ph.D.

Department of Histology, College of Dentistry, University of Illinois, Chicago, Ill.

There are in general 2 types of growth; a) multiplicative growth as a result of cell-division, and b) appositional growth or growth by addition as a result of cellular secretion (Huxley, 1932). In either case, growth is the result of cellular activity. The analysis of such cellular activity offers a fertile field for investigations in the cellular dynamics of growth.

Growth Potential of a Cell. The growth potential of a cell may be defined as the amount of growth work which a given cell is capable of performing. The growth potential can be measured by the result of that cell's activity (the total number of cells or amount of tissue matrix). The amount of growth work is in turn the product of the rate of cellular activity (division or secretion) and the time during which the cell was active (its functional life span).

$$\text{Total amount of growth work} = \text{growth potential}$$
$$\text{Growth potential} = \text{rate} \times \text{time of cellular activity}$$
$$\text{G. P.} = \text{R} \times \text{T}$$

Appositional growth is a particular form of cellular activity wherein a non-vital cellular secretion is deposited layer upon layer in the form of a tissue matrix. It differs from other, more fluid types of cellular secretion only in so far as the product of the formative cells of the hard tissues (bone, cementum, dentin and enamel) is more permanent and therefore can more readily be observed and measured after the completion of that cell's activity.

REVIEW OF LITERATURE

Enamel and Dentin Formation. For a discussion of the histophysiology and cellular dynamics of enamel and dentin formation the reader is referred to Schour and Massler (1940).

Length of Enamel Rod and Dentinal Tubule. The lengths of the enamel rods or dentinal tubules have not, to our knowledge, been accurately determined. However, they have been grossly estimated by Black (1897), Bronner (1930), Szabo (1900), Trueb (1909) and Wagner (1927).

Rates of Growth. The rates and gradients of appositional growth are easily obtained by vital injections of alizarine red S, porphyrin, purpurin, trypan blue

[1] Received for publication August 7, 1945 and revised by authors March 11, 1946.

and similar vital dyes or sodium fluoride. These injections demarcate clearly the matrix that is being deposited and calcified at the time of the injection with a sharp and characteristic line. The method of vital staining using sodium fluoride and alizarine red S has been successfully employed to determine the rates and gradients of appositional growth in the enamel, dentin and bone of various species by many investigators.

Appositional Life Span of Enamel and Dentin-Forming Cells. The appositional life span of the enamel- and dentin-forming cells has not, to our knowledge, been reported except in a preliminary abstract by Massler and Schour (1941).

METHODS AND MATERIALS

Measurement of the Growth Potential of the Ameloblasts and Odontoblasts. The final normal length (L) of an enamel rod or a dentinal fibril was taken to represent the amount of work which a given ameloblast or odontoblast was capable of performing, and therefore indicative of the growth potential (G.P.) of that cell. At any moment the length of the enamel rod or dentinal tubule (l) equals the distance traversed by the formative cell and is the product of the rate of apposition (r) and the number of days of activity (t). Similarly the final length of the enamel rod or dentinal tubule (L) is a measure of the growth potential of a given cell and is the product of the average rate of apposition (R) and the appositional life span of the formative cell (T).

$$L = G.P. = R \times T$$

Rates and Gradients of Growth of Enamel and Dentin. The material utilized in previous studies on the rates and gradients of growth of enamel and dentin was reviewed and the results used in this study (Schour and Poncher, 1937; Schour and Massler, 1937). In addition, the unreported data obtained from the teeth of two additional hydrocephalic infants who had been injected with sodium fluoride and alizarine red S were included.

Appositional Life Span of the Ameloblasts and Odontoblasts. The appositional life span of the formative cells of the enamel and dentin was obtained by dividing the final normal length of a given enamel rod or dentinal tubule by its characteristic daily rate of apposition.

Since one enamel globule is deposited each day (Asper, 1916, Schour and Massler, 1940), the life span of the enamel-forming cells was checked by counting the number of globules comprising a given enamel rod in representative teeth.

FINDINGS

Growth Potential of the Formative Cells. In the enamel, the lengths of the enamel rods graded from a maximum over the growth center to a minimum at the cemento-enamel junction. In the deciduous anterior teeth, this gradient was much less apparent than in any of the other teeth. This is further evidenced by the fact that in the deciduous anterior teeth, the surface of the enamel paralleled

the dentino-enamel junction for two-thirds of the distance between the cuspal tip and the cemento-enamel junction.

The lengths of the dentinal tubules are more uniform in length and therefore showed a much lower gradient than did the enamel rods (Table II).

Rates and Gradients of Appositional Growth. The rates and gradients of appositional growth in the enamel and dentin of the deciduous and permanent teeth are tabulated in Tables I and II. A distinct gradient of growth exists in both the enamel and the dentin. The rate is at a maxmium at the growth center and decreases smoothly as one proceeds away from it in an apical and in a pulpal direc-

TABLE I

The maximal growth potential of the ameloblasts in human teeth

	AVERAGE LENGTH OF ENAMEL ROD AT GROWTH CENTER	AVERAGE DAILY RATE OF APPOSITION	AVERAGE LIFE SPAN OF AMELOBLASTS
	μ	μ	days
Maxillary Teeth			
Decid. central incisor	640	8.0	80
Decid. lateral incisor	540	7.5	72
Decid. cuspid	720	6.0	120
Decid. first molar	935	5.5	170
Decid. second molar	1100	5.5	200
Perm. first molar	1600	4.5	355
Mandibular Teeth			
Decid. central incisor	520	8.0	65
Decid. lateral incisor	525	7.5	70
Decid. cuspid	720	6.0	120
Decid. first molar	890	5.5	160
Decid. second molar	1045	5.5	190
Perm. first molar	1600	4.5	355

The above data are based upon 250 micrometer measurements. Because no definite landmarks could be established on the enamel surface or dentino-enamel junction between the cuspal tip and the dentino-cemental junction, no other measurements of the length of the enamel rods are included.

All measurements are subject to a variation of not more than 10%.

tion. There is thus a locus gradient. There is also a decrease in the rate with the increase of age of the formative cell (age gradient).

Appositional Life Span of the Formative Cells. The life span of the formative cells is computed in Tables I and II. In the enamel, the cells have a maximal functional life span over the growth center which grades to 0 at the cemento-enamel junction.

In contrast to the ameloblasts, the life span of the formative cells of the dentin tends to reach a constant average figure in a given tooth (Table II). The active life span differs in the different teeth but grades regularly in an antero-posterior direction (Table II).

TABLE II

The growth potential of the dentin-forming cells in human teeth

	DECID. CENTRAL INCISOR			DECID. LATERAL INCISOR			DECID. CUSPID			DECID. FIRST MOLAR			DECID. SECOND MOLAR			PERM. FIRST MOLAR		
	Average length of dentinal tubule	Rate of apposition	Approximate life span of dentin-forming cells	Average length of dentinal tubule	Rate of apposition	Approximate life span of dentin-forming cells	Average length of dentinal tubule	Rate of apposition	Approx life span of dentin-forming cells	Average length of dentinal tubule	Rate of apposition	Approx. life span of dentin-forming cells	Average length of dentinal tubule	Rate of apposition	Approx life span of dentin-forming cells	Average length of dentinal tubule	Rate of apposition	Approx. life span of dentin-forming cells
	μ	μ/24 hrs.	days	μ	μ/24 hrs.	days	μ	μ/24 hrs.	days	μ	μ/24 hrs.	days	μ	μ/24 hrs.	days	μ	μ/24 hrs.	days
Maxillary teeth																		
At the growth center....	2520	8	330	2250	7.5	315	2610	6.0	450	2393	5.5	450	3220	5.5	600	3375	4.5	750
Middle third of crown...	1890	6	330	1650	5.5	315	1958	4.5	450	1740	4.0	450	2400	4.0	600	2625	3.5	750
Gingival third of crown..	1578	5	330	1350	4.5	315	1800	4.0	450	1575	3.5	450	2100	3.5	600	2250	3.0	750
At the cemento-enamel junction.....	1485	4.5	330	1260	4.0	315	1575	3.5	450	1350	3.0	450	1800	3.0	600			
Cervical third of root....	1155	3.5	330	945	3.0	315	1125	2.5	450	900	2.0	450	1200	2.0	600			
Middle third of root.....	990	3.0	330	787	2.5	315	900	2.0	450									
Apical third of root.....	825	2.5	330	630	2.0	315	900	2.0	450									
Mandibular teeth																		
At the growth center....	2280	8	300	2140	7.5	300	2310	6.0	400	2670	5.5	500	3220	5.5	600	3375	4.5	750
Middle third of crown...	1710	6	300	1670	5.5	300	1735	4.5	400	1940	4.0	500	2400	4.0	600	2625	3.5	750
Gingival third of crown..	1425	5	300	1280	4.5	300	1600	4.0	400	1750	3.5	500	2100	3.5	600	2250	3.0	750
At the cemento-enamel junction.....	1350	4.5	300	1200	4.0	300	1400	3.5	400	1500	3.0	500	1800	3.0	600			
Cervical third of root....	1050	3.5	300	900	3.0	300	1000	2.5	400	1000	2.0	500	1200	2.0	600			
Middle third of root.....	900	3.0	300	750	2.5	300	800	2.0	400									
Apical third of root.....	750	2.5	300	600	2.0	300	800	2.0	400									

The above data are based upon 1000 micrometer measurements. When the measurements cross the neonatal ring (above the dotted line in the above table) 15 days is added to the calculated life span of the dentin-forming cells to correct for the neonatal arrest in growth.

Measurements below the apical third of the root are omitted since in the sections studied, the root was either not completed or was resorbed. Data for the remaining permanent teeth are omitted since the exact rate of dentin apposition in these teeth in not yet known.

All measurements are subject to a variation of not more than 10%.

DISCUSSION

It is apparent that the growth potential and life span of the formative cells of the enamel and dentin play a significant role in the external morphology of the enamel-covered crown and the internal morphology of the dentin-covered pulp. The size and shape of the tooth is determined primarily by the size and shape of the dentino-enamel and dentino-cemental junctions which are the base from which the appositional activity of the cells begins. The morphogenesis of the crown is thus determined by the underlying dentino-enamel junction and the appositional life span of the ameloblasts which grades characteristically from a maximum over each growth center to a minimun at the cemento-enamel junction. The morphogenesis of the pulp and the parallelism of the primary pulpal outline to the dentin surface becomes readily understandable in the light of the constancy of the appositional life span of the odontoblasts.

Disturbances in the external and internal morphology of the teeth may result when the appositional life span of the formative cells is affected by disease or experimental interferences. From this point of view, enamel hypoplasia is the result of a shortened life span of the ameloblasts. Similarly, pulpal inclusions or pulpal horns (which may be regarded as counterparts of deep pits and fissures in the enamel) are the result of a failure of the dentin-forming cells to express their full growth potentiality (Schour and Massler, 1940).

The reduced size of the crowns in amelogenesis imperfecta (Weinmann, 1945) is probably a result of a hereditary disturbance in the growth potential of the ameloblasts rather than a disturbance in the dentino-enamel junction. On the other hand, the screw-driver appearance of the Hutchinson's incisor is the result of a disturbance in morphodifferentiation of the dentino-enamel junction while the growth potential and life span of the ameloblasts appear to be unaltered.

An interesting experimental interference with the expression of the growth potential of the dentin-forming cells can be observed in vitamin A deficiency. The daily rate of apposition of the enamel-covered dentin is increased from a normal daily rate of 16 μ to as high as 20 μ, while the daily appositional rate of the cementum-covered dentin is reduced to as low as 6 μ (Schour, Hoffman and Smith, 1941). The resultant pulpal outline is characteristically distorted. The growth potential of the tooth thus remains almost constant although its expression is distorted.

In the case of dentinogenesis imperfecta (hereditary opalescent dentin) the appositional life span of the dentin-forming cells is not limited to the normal length of time so that apposition of dentin continues unchecked till the pulp is completely obliterated. However, this occurs at the expense of the quality of the quality of the dentin which is atypical and irregular in structure (Roberts and Schour, 1939).

SUMMARY AND CONCLUSION

The life span of the ameloblasts was obtained by measuring the total length of the individual enamel rod and dividing it by the known rate of appositional activity.

The life span of the dentin-forming cells was obtained by measuring the length of the dentinal tubules and dividing it by the known rate of appositional activity.

The life span of the ameloblasts ranges from a maximum over the growth center to zero at the cemento-enamel junction (Table I). The life span of the dentin-forming cells is relatively constant for a particular tooth as shown in Table II.

The concept of the growth potential and the approximation of the appositional life span of the formative cells of the enamel and dentin aids in understanding the morphogenesis of the enamel, dentin and pulp.

REFERENCES TO LITERATURE

Asper, H., Ueber die "Braune Retzius'sche Parallelstreifung" im Schmelz der Menschlichen Zähne. *Schweiz Vrtljschr. f. Zahnheilk.*, **26**: 275, 1916.

Black, G. V., Descriptive Anatomy of the Human Teeth. 4th Ed. Philadelphia, 1897.

Bronner, F. S., Engineering Principles Applied to Class II Cavities. *J. D. Res.*, **10**: 115, 1930.

Huxley, J. S., Problems of Relative Growth. (New York, The Dial Press: 1932.)

Massler, M., and I. Schour, Growth Potential and Appositional Life Span of the Enamel and Dentin Forming Cells in the Human Deciduous Teeth and First Permanent Molars. *J. D. Res.*, **20**: 282, 1941.

Roberts, E., and I. Schour, Hereditary Opalescent Dentin (Dentino-Genesis Imperfecta). *Am. J. Orthod. & O. S.*, **25**: 267, 1939.

Schour, I., and M. Massler, The Rate and Gradient of Growth in Human Deciduous Teeth with Special Reference to the Neonatal Ring. *J. D. Res.*, **16**: 349–350, Aug. 1937.

Schour, I., and M. Massler, Studies in Tooth Development. The Growth Pattern of the Human Teeth. *J. A. D. A.*, **27**: 1778, 1918, 1940.

Schour, I., and H. G. Poncher, The Rate of Apposition of Human Enamel and Dentin as Measured by the Effects of Acute Fluorosis. *Am. J. Dis. Children*, **54**: 757, 1937.

Schour, I., M. M. Hoffman and M. C. Smith, Changes in the Incisor Teeth of Albino Rats with Vitamin A Deficiency and the Effects of Replacement Therapy. *Am. J. Path.*, **17**: 529, 1941.

Szabo, J., Die Grössenverhältnisse des Cavum Pulpae nach Altersstufen. *Viertelsjahrsschr. fur Zahnheilk.*, **16**: 12, 1900.

Trueb, K., Grössenverhältnisse des Cavum Pulpae nach Altersstufen. *Dtsch. Monatsschr. f. Zahnhlk.*, **27**: 401, 1909.

Wagner, F., Untersuchungen über die Grössenverhältnisse des Cavum Pulpae und seiner Wände. *Dtsch. Mschr. Zahnheilk.*, **45**: 1027, 1927.

Weinmann, J. P., J. F. Svoboda and R. W. Woods. Hereditary Disturbances of Enamel Formation and Calcification. *J. A. D. A.*, **32**: 397, 1945.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Proceedings of the Twenty-fourth General Meeting[1]

Continental Hotel, Kansas City, Missouri

March 16 and 17, 1946

Compiled by Hamilton B. G. Robinson, *Editor*[2]

Ohio State University, Columbus, Ohio

I. Inaugural address. Samuel W. Chase, Ph.D., (Western Reserve University, Cleveland, Ohio) 151
II. Index of participants and sequence numerals for corresponding abstracts 155
III. First Session: Morning, March 16; abstracts 1–6 155
IV. Second Session: Afternoon, March 16 (joint program with materials section); abstracts 7–10 158
V. Third Session: Evening, March 16
A. Materials section; abstracts 11–16 159
B. General session; abstracts 17–25 161
VI. Fourth Session: Morning, March 17; abstracts 26–36 165
VII. Fifth Session: Afternoon, March 17; abstracts 37–45 169
VIII. Sixth Session: Evening, March 17; Annual Dinner; abstracts 46–49 173
IX. Abstracts of papers read by title; 50–86 173
X. Executive proceedings and register of newly elected officers and members 187

I. Dental Research in the Near Future. Inaugural Address

Samuel W. Chase, Ph.D., Western Reserve University, Cleveland, Ohio

My remarks this evening are on a subject to which we have all given considerable thought, the future of dental research in this post-war period. Within the history of this organization research in dentistry has grown from little more than a sapling to a sturdy tree bearing a quite respectable annual yield. If we wish to improve our tree we should obviously strive to improve the quality of the fruit it will bear in future seasons. If my suggestions for improvement seem to some of you to have a slightly disagreeable odor, please bear with it in the knowledge that fertilizer does have quite an odor, yet is an excellent aid in promoting the welfare of our tree.

We have just been through a group of years which have tried us sorely. All of us have suffered from the war whether we have been in the armed services or not.

[1] The sessions of the International Association for Dental Research preceded, by agreement, those of the American Association of Dental Schools in the same hotel, March 18, 19, and 20. The annual dinner was combined with that of Omicron Kappa Upsilon and the newly elected officers of both groups were installed at that meeting.

[2] The Editor wishes to express his appreciation to the General Secretary, E. H. Hatton, for his cooperation in the compiling of these proceedings.

A fortunate few have been able to give a reasonably adequate part of their time and energy to research, but by far the majority of us have been so overburdened by the added loads imposed by accelerated programs and depleted staffs that research programs have had to be curtailed or postponed.

Now, as we enter this also trying period of reconversion, let us look at the prospects for research in denistry in the near future. By taking cognizance of the inadequacies of the past and present may we not, perhaps, lay plans for a more adequate and more efficient research program in the years ahead?

Good research requires ample time and a free mind. Only the rare individual can pursue investigations in disconnected moments snatched from a schedule heavily loaded with teaching, administrative duties, or the demands of practice. With our load practically doubled during the war years, but little time and energy were left for other affairs. When, during those years we were badgered and bedevilled also by the petty, annoying, and time consuming demands of regimentation and bureaucracy, we were rarely in the proper mood for constructive, productive work.

Now we have been freed from some of this extra demand on our time, but not from all of it. Concerning a heavy load of teaching or other duties, it is rumored that the administrative officers of certain of our institutions of learning, having discovered during the period of emergency how heavy a burden the human body and mind can bear, and having ever an eye for a bargain, are taking advantage of that discovery by continuing to make heavy demands on their faculties to the detriment of both teaching and research. Let us hope that this is no more than rumor. Or, if there is more than rumor in it, let us hope that such administrators learn early that not every bargain is cheap.

Concerning the demands of regimentation and bureaucracy, we have been freed from much of the annoyance and from many of the symbols of regimentation, but bureaucracy is still with us. It is going to be as difficult to get rid of as was Sinbad's Old Man of the Sea. Let us do all in our power to encourage and support the recent efforts of a few of our Congressmen to come to the aid of poor Sinbad. Their small spark of rebellion must be fanned to a hot flame of nation-wide revolt against our strangling, ever more burdensome Old Man Bureaucracy before we can lighten ourselves of his load.

It was mentioned above that the productive investigator must have a free mind. By this I mean a mind as free as possible from worry and discontent. Since a large share of the worry and discontent of most of us stems from financial inadequacy, may we not hope that means can be found to narrow the wide gap between the reward for mental activity and that for manual labor? "Pity the plight of the white-collar man" is a much voiced theme, but it is, alas, but a feeble voice in the wilderness. The cost of living has increased just as much for the white-collar man as for the wielder of a wrench, but the white-collar man has neither been willing nor able to descend to the level of those groups which are willing to jeopardize the welfare of our whole nation for a long time to come in their exploitation of the present precarious situation for the attainment of their greedy ends.

Many of us see hope of easement from this situation in the projected aids to scientific investigation now being so actively discussed by Congress. Some others are not so optimistic about the prospects for this National Science Foundation. For example, a recent article in *Science* by Professor Germann of the University of Colorado questions the benefit of such a program to the individuals most in need of benefit, and Doctor Jewett, President of the National Academy of Sciences, in a recent Sigma Xi National Lecture, made a gloomy prediction of our inability to keep such a program out of the hands of the politicians. If these men are right, governmental aid for a research project might well turn out to be a mixed blessing.

Another aspect of the future of dental research is of present interest. In recent months we have heard much about the loss of a large part of a generation of young scientists. Several leaders in the scientific world have sounded pessimistic notes on this theme. It is true that our Selective Service largely ignored the need for replacement of personnel in scientific fields, particularly in the physical sciences; it is true that many promising young scientists have been casualties in the war; and it is true that many others have been sidetracked by interruption of their training and by diversion into fields other than those of scientific research.

On the other hand, it is also true, particularly in the fields of medicine and dentistry, that many men who would not otherwise have been able to finance their training have been able to receive that training through the lavish provisions of the A. S. T. P. and V-12. Many more have had their thoughts turned to the preservation of life and health as a direct result of their observations and experiences during the war. Many of these are receiving or will receive training with the aid of the "G. I. Bill" and the Veterans' Administration.

Out of this large pool of men it seems reasonable to expect that a normal complement will have in them that peculiar combination of overdeveloped bump of curiosity, keen analytical sense, and capacity for persistence in the face of repeated failure which makes the true investigator.

For these reasons I am not at all pessimistic about the future as concerns the number and quality of a new supply of reserach men.

Perhaps a more disturbing situation is the dilution of our staffs of investigators in our universities due to the rapid exodus of trained men into industrial and commercial organizations. The number of fine teachers and able investigators who are deserting inadequately recompensed university positions for the vastly better financial rewards of industrial research has reached alarming proportions. In most cases replacements for these losses have been made from the bottom. A recent article on this subject contained the homely analogy of skimming off the cream of outstanding men and leaving only the skimmed milk of those who lack capacity for leadership. If we pursue this analogy further, it raises the thought that, though soured skimmed milk is undoubtedly nutritious and of some value in preparing pigs for market, a steady diet of lumpy curds and thin, watery whey is scarcely one which whets the appetite or stimulates the mental processes of the human animal. .

As another problem in connection with dental research I would like to mention the subject of forced research and particularly that evil which is a direct corollary of it, forced publication. While a few of us, including myself, are by nature so indolent and so prone to procrastination that we require a goad to keep us in productive motion, it is still true that research should not be forced. Rather it should spring from a compelling interest and an urge that cannot be denied. To require results in the form of the publication of a certain number of papers or a certain number of pages a year in order to hold a position or to ensure the continuation of a grant is to encourage the publication of too much about too little. Such practices have been only too prevalent in late years. Multiple publication, that is the publication of essentially the same data in several journals, or in thinly disguised alteration of form and title in the same journal is one method known to all of us. Padded papers, so common in Continental Europe, are another method. In these an article which could have been adequately and more forcefully treated in ten or twelve pages is expanded to close to a hundred pages, or even in some cases to the dimensions of a book. Still a third method is much employed by collaborative groups. You all know the technique. John Doe and Richard Roe publish a paper on "The Effect of Tids on Tods". Within a short time there appears a paper by Richard Roe and John Doe on "Tods as Affected by Tids". Next there is a paper by John Doe and Richard Roe entitled "The Alteration of Tods under the Influence of Tids" and, finally, a paper by Richard Roe and John Doe on "The Influence of Tids on the Alteration of Tods". By introducing another factor or two and adding co-authors one can figure mathematically the prodigious number of combinations and permutations possible. The net result is to extend the list of papers published by each co-author, to overload certain journals in which editorial control is not too hard-boiled, to made the paths of the abstractor and librarian more stony, and to add just one item to the store of human knowledge.

Knowing what we do about the desire of those who finance some of our research programs to expect rapid and fruitful returns on their investment, one cannot be too censorious of those who resort to these techniques. Really the cure for this condition lies in the education of those in control of funds granted for research. These individuals, many of them hard-headed business men used to the methods of industry, must be convinced that good research cannot be turned out on a production schedule nor delivered on demand as is merchandise from a vending machine when the proper coins are dropped in the slot.

In closing I would like to summarize by saying that, if intelligent, adequately trained, intellectually honest men with an avid interest in the problems of denistry are chosen; if these men are paid salaries sufficient to free their minds from financial worries; if they are given ample time and facilities for efficient research on subjects of their own selection; if they are freed from the necessity of working on a production schedule; and if they publish only when they have findings which are worth publishing; then the future of dental research will take care of itself. If these rules are followed, there is but little further need for a planned program for dental research.

II. Index of Participants, and Sequence Numerals of Corresponding Abstracts

Armstrong, W. D., 3, 50; Asling, C. W., 54; Barnfield, W. F., 51, 52; Becks, H., 53, 54, 55, 59, 60; Berg, M., 40, 41; Bibby, B. G., 19, 56, 70, 71; Bodecker, C. F., 57, 58, 72; Boelsche, R., 33; Boyd, J. D., 20, 21; Boyle, P. E., 2; Brodie, A. G., 32; Burrill, D. Y., 40, 41; Chase, S. W., 38; Cheyne, V. D., 20, 21; Collins, D. A., 53, 55, 59, 60; Collins, P., 8; Cook, T. J., 61; Crawford, H. M., 25; Dam, H., 65; Davidson, N. R., 2; Dummett, C. O., 1; Eisenbrandt, L., 62; Evans, H., 53, 54, 55, 59, 60; Falkenheim, M., 18; Finn, S. B., 43; Fosdick, L. S., 40, 41; Foster, W. C., 74, 74; Frisbie, H. E., 28; Gainsforth, B. L., 86; Gilda, J. E., 17; Glickman, I., 37; Gottlieb, B., 24, 25, 33, 34, 63, 64; Granados, H., 65; Hampp, E. G., 22; Harmon, I. M., 12; Hemmens, E., 45; Hillier, J., 2; Hine, M. K., 79; Hirschfeld, I., 66; Hirschfeld, L., 66; Hodge, H. C., 18; Hollenback, G. M., 10; Ireland, R. L., 35; Irons, W. G., 67, 68; Karlan, F. R., 69; Karshan, M., 69; Keyes, P. H., 26, 27; Kitchin, P. C., 29; Korns, R., 43; Labunsky, B., 56; Lawyer, A., 70, 71; Lefkowitz, W., 57, 72; Leiscester, H. M., 30; Leonard, H. J., 69; Likins, R. C., 27; McClendon, J. F., 73, 74, 75; McClure, F. J., 76; McKelleget, M., 56; McKinnon, I., 19; Mason, K. E., 65; Moore, A. W., 4; Moore, T. E., 7; Nuckolls, J., 23; Orban, B., 36; Ortiz, M., 31; Permar, D., 29; Phillips, R. W., 77, 78, 79; Pinkerton, D., 44; Pomes, C. E., 16; Ray, K. W., 11; Revesz, J., 38; Robinson, H. B. G., 29; Rosenstein, P. F., 80; Ruzicka, S. J., 76; Scott, R., 81; Shapiro, H., 72; Shell, J., 14; Sicher, H., 6; Simpson, M., 53, 54, 55, 59, 60; Slack, F. A., Jr., 15; Stephan, R. M., 45; Swanson, W. F., 82; Tenenbaum, B., 69; Thomas, B. O. A., 31; Vissotzky, L., 85; Volker, J. F., 44; Wah Leung, S., 39, 81; Wainwright, W. W., 83; Watts, C., 13; Weinmann, J. P., 5; Wellensiek, E. K., 84; Weston, P. E., 42; Wise, E. M., 9; Younger, H. B., 24; Zander, H. A., 56, 71, 85.

III. First Session, Morning, March 16; Abstracts 1–6

1. The relationship between physiologic pigmentation in the oral and cutaneous tissues of the negro. *Clifton O. Dummett, B.Sc., D.D.S., M.S.D., Meharry Medical College, School of Dentistry, Nashville, Tenn.* Pigmentation of oral and cutaneous tissues is often considered an important diagnostic factor in pathologic conditions, however, pigmentation of these tissues also occurs in persons who on thorough examination show no associated pathologic changes. This is especially true of Negroes, although physiologic pigmentation is not limited to any one race. It has been reported in many Caucasians. About 600 Negroes of varying shades of complexion were examined to find relationship of oral to cutaneous pigmentation. The gingivae, buccal mucous membranes, tongue, hard palate and lips were examined and observations were made of the type, character and region of pigmentation. The general complexion of the skin in the lower half of the face (from the tip and base of the nose to the mentum) was used as a basis of comparison with the color of the oral tissues. Because of the wide variation in skin color of Negroes, it was decided to use 4 complexion classifications and exclude those cases in which a possible con-

flict in color determination might occur: (1) Very dark. (2) Brown. (3) Light brown. (4) Fair. In the cases examined, gingival tissue pigmentation was directly proportional to skin pigmentation in a large majority of cases, that is, those Negroes of very dark and brown complexions showed considerable oral pigmentation while Negroes of light brown and fair complexions showed much lessened amounts of pigmentation. However, the number of cases of very dark Negroes with no clinical evidences of pigmentation, were enough to question the unvarying constancy of association between oral and cutaneous pigmentation.

2. PRELIMINARY OBSERVATIONS OF THE ENAMEL OF HUMAN AND GUINEA PIG TEETH USING THE ELECTRON MICROSCOPE. *Paul E. Boyle, D.M.D., James Hillier, Ph.D., Norman R. Davidson, Ph.D., University of Pennsylvania, Philadelphia, Pa.* Unerupted central incisor teeth of a full term human fetus and incisor teeth of guinea pigs were used as sources of enamel. Samples of soft, relatively uncalcified enamel and of successively harder enamel were used up to point where enamel could no longer be scraped or easily broken from tooth surface. No fully calcified, mature enamel was examined. Specimens were dispersed in colloidin using convex surface of a watch glass with fair amount of digital pressure and then examined in the electron microscope. The technique may have been sufficiently severe so that beside breaking the enamel rods and interprismatic substance into fundamental crystallites it is possible that some of the crystallites were broken into still smaller particles. Micrographs provide visible confirmation of the ideas set forth by Thewlis (*Proc. Phys. Soc.* **51:** 992, 1939) concerning structure of enamel prisms. Each prism must contain a "multitude of enamel crystallites", however, the crystallites appear to be about 40 to 60 millimicrons wide rather than 200 to 500 as he suggested. In some aggregates all crystallites are aligned along a single axis, in others, especially larger masses, there are regions of clearly different orientation. There are amorphous masses of material which have not been identified but which possibly may be organic matrix of enamel. A striking feature of micrographs is distinction between the long crystallites of the more mature guinea pig enamel and much shorter crystallites of guinea pig enamel nearer formative end of tooth and rather similar short crystallites of human enamel. Good electron diffraction patterns were obtained from dispersions of more mature guinea pig enamel by one of us (N. R. D.). Quantitative interpretation of these patterns indicates that material is fluor- or hydroxyapatite. Precision of method is not sufficient to indicate whether lattice constants are closer to those for fluor- or hydroxyapatite. The ool reflections have a greater intensity relative to the general (hkl) reflections in electron diffraction patterns than is case for x-ray powder diffraction data for apatites. Relative intensities of different (hkl) lines are normal. These effects in electron diffraction pattern due to crystal orientation are in accord with impression obtained from micrographs that crystals are oriented with hexagonal axis perpendicular to the electron beam. The direct examination of pulverized enamel in electron microscope has been reported by Gerould (*J. D. Res.* **26:** 223, 1945). Appearance of crystallite units of enamel has not heretofore been demonstrated.

3. BONE GROWTH IN PARALYZED LIMBS. *W. D. Armstrong, Ph.D., M.D., University of Minnesota, Minneapolis, Minn.* To study the effect of denervation on growth of calcified tissues the nerves of the brachial plexus on the right side of young rats were severed and the length of the shadows of the humeri and radii were measured on roentgenograms taken at intervals over a period of 15 weeks. The diameters of the bones, their weight and ash weight were determined after sacrifice of the animals. It was found: 1, Growth in length of the radius was affected earlier and to a greater degree than that of the humerus. 2, Reduction of growth in diameter of the humerus was greater than the reduction of growth in length. 3, The quantity of bone and of bone salt was reduced by about 25%. It is concluded that denervation of bone interferes more with appositional growth than with epiphyseal growth.

4. AN ACCELERATED METHOD OF EMBEDDING ANATOMICAL SPECIMENS IN METHYL METHACRYLATE. (A Preliminary Report.) *Alton W. Moore, D.D.S., University of Illinois, School of Dentistry, Chicago, Ill.* A method of embedding young alizarin-injected macaque monkey skulls in hard substance that would permit making ground sections with maintanence of accurate anatomical relationships was sought. Most satisfactory material found unpolymerized methyl methacrylate monomer. Inhibitor was removed from monomer and accelerator was added by methods previously reported by other authors. Technic found most satisfactory for embedding skulls in methyl methacrylate was prepolymerization of methyl methacrylate into 2 different consistencies; 1, thin solution having consistency of glycerine, and 2, thick solution having consistency of very heavy molasses. Thin solution will keep under refrigeration indefinitely while thick solution must be used immediately after preparation. Suitable glass vessel is selected for embedding specimen which is oriented in desired position. It is completely covered with thin prepolymerized monomer and subjected to vacuum for 5 to 10 minutes and then allowed to stand at room temperature for 24 hours with loose fitting cover. Specimen is then covered to an excess of 1 inch with thick prepolymerized which will displace thin monomer and vessel is left at room temperature for 48 hours or until thick monomer is completely hardened. Thin monomer will still be fluid and should be decanted off at this time. In another 24 hours surface should be completely hardened. Final stage consists of annealing the block in an oven at 50°C., so as to prevent checking or crazing. This technic differs from those previously described only in manner of embedding. One half of a macaque monkey skull was embedded by this procedure in a block of methyl methacrylate measuring $5\frac{1}{2}''$ x $5\frac{1}{2}''$ x $2''$, the total time involved was 1 week. Relatively few bubbles were present in specimen. Value of this type of embedding in making ground sections of bone and teeth is now being investigated.

5. NUTRITIONAL AND HORMONAL CHANGES OF THE MANDIBULAR CONDYLE. *Joseph P. Weinmann, Loyola University, School of Dentistry, Chicago, Ill.* Condyles of 37 young rats were examined to determine the changes in experimental rickets and in experimental hyperparathyroidism. The lack of calcification of the condylar growth-cartilage inhibits its resorption during "endochondral" growth. The normally thin cap of cartilage at the condyle is then thickened to

such a degree that the entire condyle seems to consist of cartilage. The bone deposited during experimental rickets does not calcify and therefore is resistant to resorption. The most striking consequence is the lack of modeling resorption at the mandibular neck which is disfigured to a plump, club-shaped bone. Injections of 200 H. U. parathyroid hormone daily after 3 days lead to the well known exaggerated osteoclasis. The consecutive osteoporosis in the region of the mandibular neck may lead to spontaneous fracture. If injections of parathyroid hormone are prolonged, a process of healing sets in. One of the first signs of repair is the formation of osteophytes on the surfaces of the porotic bone. This can be regarded as an emergency reinforcement.

6. THE GROWTH OF THE MANDIBLE. *Harry Sicher, Loyola University, School of Dentistry, Chicago, Ill.* The most important sites of growth of the mandible are: condylar cartilage, tip of coronoid process, posterior border of the ramus, and free border of the alveolar process. Modeling resorption is most prominent at the anterior border of the mandibular ramus. The condylar growth-cartilage, often compared to an epiphyseal cartilage, differs from the latter in that it is covered by a layer of connective tissue. Therefore this cartilage grows interstitially and appositionally. Chondrodystrophy, which is a genetically caused inhibition of the interstitial growth of cartilage, has little influence upon the growth-cartilage of the mandible where lack of interstitial growth is compensated by appositional growth. Although it is correct that the contour of the mandibular angle does not change during growth, it has to be recognized that the inclination of the condyle to the body of the mandible changes regularly. The mandibular angle of the physical anthropologist, diminishes from birth to adulthood. The significance of this fact is, that in the first period longitudinal growth of the mandible is more pronounced than vertical growth, while in the later period these processes are reversed.

IV. SECOND SESSION, AFTERNOON, MARCH 16; ABSTRACTS 7–10

7. DEVELOPMENTS IN INVESTMENTS FOR THE CASTING OF GOLD ALLOYS AND BASE METALS. *T. E. Moore, Ransom and Randolph Company, Toledo, Ohio.* The composition, physical and chemical properties of the various investment types that are used in dentistry to make inlays and partial denture castings of gold and base metal alloys are described. The application of the dental investments and the lost wax process to industrial use for casting critical war parts of non-ferrous, ferrous and high melting chromium alloys, also are described. New investments and processes that were developed and used during the war, are included in the discussion.

8. THE USE OF BASE METAL ALLOYS IN CASTING. *Paul Collins, Austenal Laboratories, Chicago, Ill.* The development of industrial casting practices based on the lost wax method common in dentistry and the extent of the application in industry is described. The nature of the investment used for casting Vitallium type alloys for supercharger buckets and other aircraft motor parts are discussed. It is clearly indicated that practice and experience gained from dental casting practices were of value to the industrialist. In like manner it is expected that

added information gained by industrial research during recent years will be of importance in the future improvement of design, soundness of castings, and quality dental structures.

9. PHYSICAL METALLURGY OF PRECIOUS METAL ALLOYS. *E. M. Wise, The International Nickel Company, New York.* Discussion is given of the properties of the individual precious metals and the systems Au-Ag-Cu, Pd-Ag-Cu, as well as the more complicated alloys containing platinum and palladium derived from these systems, which are the basis for the alloys broadly employed in dentistry. Some discussion also is given to precious metal alloys having higher melting points than the preceding group. Attention likewise is given to some of the precipitation hardening transformations, melting behavior, etc. of dental alloys.

10. LINEAR CASTING SHRINKAGE OF GOLD AND ITS ALLOYS. *George M. Hollenback, Los Angeles, Cal.* Linear shrinkage of pure gold and its alloys was established by Coleman as being 1.25% ± 0.1%. Coleman believed the variation which might exist between pure gold and alloys came within the element of experimental error and therefore was incomputable and of little consequence. Sweeney, later, did further research on this subject, using the same equipment as Coleman and in addition another simpler apparatus. Sweeney's results coincided with those of Coleman's though it is noteworthy that with this simpler apparatus the element of experimental error was ± 0.2%. 1.25% was quite generally accepted though there never has been complete agreement as to its correctness. Shell, Lane and Scheu found values considerably different from each other, none of them agreeing with the accepted figure, 1.25%. Scheu, for the first time, clearly proved that in making of precision castings, varying degrees of compensation were required for different alloys. This phenomena had been noticed by many but Scheu was the first to demonstrate it in a scientific manner. An extensive study was made on this subject, using very accurate apparatus under carefully controlled conditions. The linear casting shrinkage of pure gold was established as 1.65% ± 0.03%. Eleven experimental and 4 commercial alloys were tested. The linear casting shrinkage of the former varied from 1.3% to 1.5%, of the latter 1.37% to 1.56%. Later studies, using other types of apparatus and methods have all agreed very closely with these values. It is very interesting to note that alloying metals have a marked effect in the reduction of shrinkage. This shrinkage is, however, not proportional to the percentage of the alloying metals. It seems highly desirable that further studies be made on the effect of the various metals used in the alloying of gold on linear casting shrinkage. Studies should be made of the effect of each alloying metal and their combined effect.

V. THIRD SESSION, EVENING, MARCH 16; A (MATERIALS GROUP); ABSTRACTS 11–16

11. EFFECTS OF HEAT TREATING AMALGAM ALLOY INGOTS BEFORE CUTTING INTO FILINGS. *K. W. Ray, General Refineries Inc., Minneapolis, Minn.* The heat treating of high silver amalgam alloy ingots before they are cut into filings,

followed by the usual anneal of the cut alloy, has pronounced effects upon the properties of the amalgam made from the filings. The temperature to which the ingots are heated, the length of time of the anneal, and the rate of cooling employed, all have an effect upon the amalgam. In general, the rate of set of the amalgam, its setting expansion, and its smoothness and plasticity can be varied over wide limits. Amalgams made slow-setting by heat treating the ingots are inclined to be low expanding and harsh, while the medium setting amalgams produced are more highly expanding and are more smooth. The variations in the properties of amalgams due to the heat treating of uncut ingots were pronounced with an alloy 70% of silver, but were absent in an alloy containing 68% silver.

12. Effect of time and temperature on polymerizatien of acrylic resins. *Ida M. Harman, Vernon-Benshoff Company, Pittsburgh, Pa.* A report on some physical properties of an acrylic resin denture base resulting from various curing techniques. Several combinations of time and temperature were used to produce specimens for comparing such properties as long-term dimensional stability and water sorption, and transverse strength. The discussion includes consideration of the importance of temperature standardization in any measuring procedure, and the effect of mass and form on the strength of an irregularly-shaped sample. A sample form was developed to simulate the irregular distribution of mass in the denture and to demonstrate the relationship between the strength of any portion of the sample and the temperature reached in that portion during polymerization. It is further concluded that the size of the cured sample from which a specimen is cut is a critical factor in specification testing for strength.

13. A study of investment expansions required for gold inlay casting. *Claude Watts, Ransom and Randolph Company, Toledo, Ohio.* This is a preliminary report giving the results of expansion tests on an investment material and includes data on thermal expansion, setting expansion, and hygroscopic expansion. A comparison of results obtained by 2 different methods of determining setting expansion is made. Consideration is given to the effect of the shape of the wax pattern on the investment expansion. Results of experimental tests on inlay castings are given correlating such factors as type of inlay gold, total investment expansion, and fit of the casting.

14. Development of silver alloys with specialized physical characteristics. *John Shell, Thomas J. Dee and Company, Chicago, Ill.* During the last 10 years, numerous rather complicated and unusual alloys of silver have been employed for specific industrial uses. The requirements may vary from a high electrical conductivity to a high tensile strength. Silver as a basis for such alloys has been found to contribute remarkable properties not generally attainable by the use of any other metal.

15. A study of dimensional change in the methacrylate resin bases. *Fred A. Slack, Jr., H. D. Justi & Son, Inc., Philadelphia, Pa.* This paper describes the change in dimensions of methacrylate resins from 212°F. polymerization temperature to room temperature. It has been found that a change in temperature is significant in the fit of dentures or test patterns.

16. RESEARCH ON ALGINATE IMPRESSION MATERIALS. *C. E. Pomes, Northwestern University School of Dentistry, Chicago, Ill.* Eight alginate impression materials have been investigated for physical characteristics related to gelation, compressive properties and accuracy of reproduction. The gelation time decreases with increase in the temperature of the gauging water, but is independent of the spatulation time with the limits employed. The compressive strength and elasticity are dependent on the spatulation time and, in general, they increase for 4 to 16 minutes after gelation, depending upon the type of material. The accuracy of reproduction compares favorably with that of the agar impression materials.

V. THIRD SESSION, EVENING, MARCH 16, B. ABSTRACTS 17–25

17. A STUDY OF THE MANLY-HODGE SEPARATION METHOD AS APPLIED TO DENTAL TISSUES OF THE RAT AND HAMSTER. *J. E. Gilda, University of Rochester, School of Dentistry and Medicine, Rochester, N. Y.* Enamel and dentin of pooled molar crowns from 1 or several animals were separated using the method of Manly and Hodge. The teeth were ground to pass one of a number of screens (60, 100, 160, or 200 mesh). An acetone-bromoform solution of d = 2.70 was used. The separation tubes were brought to 2,200 RPM in 2 minutes and maintained at this speed for 2 to 5 minutes. Percentage composition of fractions was determined by count of 1000 particles. Purity of fractions powdered to pass a 60 mesh screen was less than previously reported for separations of human tissues. Purity rose as teeth were ground finer. Average percentage of particles powdered to 200 mesh were: Enamel fraction—enamel, 94.3; dentin, 1.2; junction—particles, 4.5; dentin fraction—dentin, 82.9; enamel, 14.6; junction—particles, 2.5.

18. AN OBSERVATION ON THE MECHANISM OF FLUORIDE ADSORPTION USING RADIOACTIVE PHOSPHORUS. *Marlene Falkenheim and Harold C. Hodge, The University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.* Of radioactive phosphate adsorbed on bone in 1 hour, 40% remain on the bone when it is exposed to a non-radioactive phosphate solution of the same concentration for 1 hour. When the bone marked with radioactive phosphate is exposed to a solution of sodium fluoride of the same molar concentration, 85% of the radiophosphate remain on the bone after 1 hour. The same amount of radiophosphate is desorbed when distilled water is used in place of the NaF solution. If fluoride were replacing phosphate in the lattice of the apatite crystal, a more marked desorption of radiophosphate would be expected. This is evidence that phosphate is not replaced by fluoride.

19. EFFECT OF VARIOUS AGENTS ON ENAMEL SOLUBILITY. *B. G. Bibby and Irene McKinnon, Tufts College Dental School, Boston, Mass.* Solutions of salts (nitrates, chlorides, sulphates and acetates) of various metals (0.1% in pH 4 buffer) were exposed to 100 mg. samples of powdered enamel for 20 min. to determine their effects on solubility during a subsequent 20 min. treatment with pH 4 sodium acetate-acetic acid buffer. The lowest solubility followed treatment with lead (9.4%); in the next lowest group were lithium (20.5%), iron,

gold, cadmium, copper, uranium and cobalt; then iron (ferrous), zinc strontium, molybdenum and chromium; and then silver, nickel, thallium, mercury and magnesium, which gave essentially the same solubility figures as treatment with water (32.7%). Reagents in citrate buffers were slightly more and in lactate buffers slightly less effective than in acetate buffers. Treatment with manganese sulphate increased the solubility to 46.2%. Exposure to various fluorides revealed the lowest solubility after the use of lead fluoride (3.9%) with increases in the following order: CnF_2, SrF_2, NaF, NH_4F, KF, ZnF_2, BaF_2, AgF, FeF_3, LiF_2, CaF_2. Solubility reductions produced by sodium oxalate suggested that fruit and vegetable extracts might have kindred effects. Of those tested only rhubarb juice (20.5%) seemed to produce any definite reductions of solubility. The lowest solubilities yet noted followed treatment with lead and indium fluoborates. Values given are not absolute since the results have not been corrected for weight accretions resulting from reagent treatments.

20. SERIAL COURSE OF DENTAL CARIES IN CHILDREN RECEIVING RECURRENT DENTAL CARE. *Julian D. Boyd, M.D., and Virgil D. Cheyne, D.D.S., Ph.D. State University of Iowa, Iowa City, Ia.* Serial dental records from files of Children's Clinics of 60 boys and 41 girls have been reviewed, to determine course of tooth decay in presumably representative child population. Each child included had been examined at least 6 times during period of not less than 3 years. At each visit necessary reparative dental therapy had been performed in competent manner. Techniques of examination and of recording had been constant. Despite recurrent dental care, caries had progressed practically without exception. In 75% of children average annual increment of newly carious tooth surfaces was not less than $2\frac{1}{4}$, median value was 3.8 surfaces. In contrast to this evidence of progressive tooth destruction, during course of study only 1 permanent tooth was lost because of decay among all. Much individual variation in caries progression was noted; not only among subjects, but in course of segments of observation. Children with least amount of caries at initial examinations showed greater inconstancy as in subsequent caries pattern than did those with high initial incidence. Those whose initial caries scores placed them in middle $\frac{1}{2}$ of group showed greater tendency to remain in that position. Nature of study did not permit much analysis of factors which might have contributed to nature or variability of dental response. Most of subjects had always lived in Iowa City, where drinking water does not contain significant amounts of fluorine. Practically, it may be concluded that recurrent dental therapy does not of itself prevent development of tooth decay. Low incidence of tooth loss speaks for protective effect of reparative dental treatment, however. From viewpoint of dental research study exphasized unreliability of data based on short-term periods of observations as evidence of efficacy or inefficacy or specific caries-affecting agents. Data indicate that it may be more profitable to employ as experimental subjects those children whose past caries experience places them in median group rather than to choose random sample of subjects without regard for earlier caries course.

21. INCONSTANCY OF CARIES IN AN AREA OF ENDEMIC FLUOROSIS. *Julian D.*

Boyd, M.D., and Virgil D. Cheyne, D.D.S., Ph.D. Colleges of Medicine and of Dentistry, State University of Iowa, Iowa City, Ia. The authors hoped to gain quantitative data as to amount and constancy of caries reduction in an area where mottled enamel is endemic. They have studied in detail incidence and pattern of caries in mouths of 85 high school pupils from Ankeny, Iowa, where many of wells provide water of high fluorine content, and likelihood that residents of that area will not ingest appreciable amounts of fluoride is small. Until 1936, municipal water supply contained more than 6 ppm. of fluorine; since content has been 1 ppm. Rural wells have been reported to contain from 0.4 to 6.4 ppm. of fluorine. Mottling of enamel is commonly observed among children born and raised in locality. Two-thirds of high school students examined had mottled enamel. Only 2 children (boys) were entirely free from caries lesions, and each had minimal evidence of mottling. Average incidence of caries was 11.4 DMF tooth surfaces for each mouth. Ratio of DMF surfaces to DMF teeth was 1½:1. In accompanying table, caries scores for 3 subgroups of children are compared. Values for subgroup with mottling are similar to those

	NUMBER	AVERAGE AGE	DMF SURFACES		DMF TEETH	RATIO BETWEEN DMF SURFACES, DMF TEETH
			Ave.	Range for middle ½	Ave.	
Subjects with no mottling of enamel	35	15.9	14.9	7–23	9.6	1.6:1
Subjects with mottling	50	15.5	9.0	4–12	6.7	1.3:1
Subjects with mottling whose water supply for at least the past 7 yrs. has contained noteable amounts of fluorine	14	15.0	8.0	5–11	6–1	1.3:1

for whole group with mottling. Difference between means of those with and without mottling is highly significant. Obviously there is high correlation between presence of mottling and the average caries score, but, 1, average caries score for children with mottling is notably high and 2, there is wide variance in caries scores among those with fluorosed enamel, even among those who are known to have maintained ingestion of fluoride at presumable protective level. In view of commonness of high fluorine levels in water of this community, one might expect that even those subjects with no mottling of enamel would show appreciable lessened caries incidence, if fluoride ingestion after eruption of permanent teeth may lessen caries incidence. The data reflect no such trend. One may conclude that despite some diminution of caries in subjects with mottled enamel, fluoride ingestion has not prevented significant caries incidence in such subjects. Further, there is little or nothing to indicate that fluoride ingestion subsequent to eruption of permanent teeth has offered noteworthy protection against caries initiation or advance.

22. AGGLUTINATION STUDIES OF THE SMALLER ORAL TREPONEMATA, BORRELIA VINCENTII AND CULTURED STRAINS OF TREPONEMA PALLIDUM. *Edward G. Hampp, D.D.S., M.S., National Institute of Health, Bethesda, Md.* The oral

spirochetes have been known and studied for many years yet no investigations have been reported in regard to serological procedures. In this study the agglutination test was selected as the first method of approach to the problem and the microscopic test described by Schuffner and Mochtar for leptospirosis icterohaemorrhagica was utilized. Eight strains of the smaller oral *Treponemata*, 3 strains of *Borrelia vincentii* and 4 cultured strains of *Treponema pallidum* were used in these investigations. Attempts at surface cultivation of these spirochetes for the purpose of obtaining organisms for immunizing animals was unsuccessful. A liquid medium containing 0.1% agar and 2.5% mucin was developed for mass cultivation of the spirochetes and the whole cultures proved suitable for intravenous inoculation of rabbits. However, this method of cultivation was not feasible for supplying antigen for the agglutination test, consequently a method was devised for growing spirochetes in a clear liquid medium for this purpose. Experiments were carried on with each organism to determine the dosage and number of intravenous inoculations necessary to develop agglutinins of high titre in rabbits. It was found that the oral spirochetes as well as the *T. pallidum* strains produced agglutination titres of 1:1000 in rabbits which had received 2 intravenous inoculations of specific organism. With an increasing number of injections it was found that the titres also increased. After 8 intravenous inoculations positive agglutinations with homologous sera were obtained in dilutions of 1:10,000 and in 3 instances titres of 1:1,000,000 were attained.

23. A PRELIMINARY REPORT ON THE BACTERIOLOGY OF THE CARIOUS LESION IN THE DENTIN: A STUDY OF THE BEHAVIOR OF MICROORGANISMS TAKEN FROM THE DENTIN BENEATH THE UNBROKEN ENAMEL SURFACE. *James Nicholls, University of California, College of Dentistry, San Francisco, Cal.* Structures closely resembling bacteria are present in the histological sections of the organic matrix of carious teeth. In the deeper enamel and in the dentinal tubules these structures are uniformly round or oval and look like cocci. In more superficial areas they vary from short rods to long thread-like bodies. We have attempted to recover and cultivate these forms from carious areas of dentin beneath unbroken enamel surfaces, to isolate and identify the organisms, and to study their metabolic activities. A variety of media was used under both aerobic and anaerobic conditions. These cultures showed many different types of organisms, several of which corresponded morphologically to the types observed in the histological sections of the carious enamel and dentin matrix. The behavior of these organisms, when cultured under various conditions, corresponded roughly to the published descriptions of other investigators. These preliminary studies suggest a broad overlapping group of highly pleomorphic types which indicates the need for further study and a clarification of existing classifications.

24. HYPERCALCIFIED DENTIN PREVENTS SPREAD OF CARIES AND DAMAGE TO THE PULP. *H. B. Younger and B. Gottlieb, Baylor University, College of Dentistry, Dallas, Tex.* Some untreated cavities do not extend to the pulp for years; others reach the pulp in a short time. Where acidogenic microorganisms are present, decalcification by acid may produce an opaque hypercalcification in the periphery through filling in the dentinal tubuli with calcium salts. This hyper-

calcified barrier prevents spreading of microorganisms. Spreading of microorganisms stops, yellow pigmentation fills in and the progress of caries is arrested. In other cases, no barrier is formed and caries progresses to the pulp. In cavity preparation we should first find out with the hand excavator what kind of cavity confronts us. In the cavity with a hypercalcified barrier we hit a hard, hypercalcified wall. In this case we proceed with preparation and filling with comfort. If we find that no barrier exists toward the pulp, we must stop after removing the necrotic dentin, impregnate with a strongly acting silver nitrate compound, make a temporary filling, and proceed after no less than 3 months with cavity preparation and restoration. Usually we will find a hardening of the previously soft dentin. This procedure is safe if no spontaneous pain has indicated damage to the pulp. If there has been spontaneous pain we can not be sure that the pulp will recover. Before starting final cavity preparation we should find out if the pulp is still living.

25. VARIOUS DEGREES OF DEVELOPMENT OF HYPERCALCIFIED BARRIERS IN THE DENTIN. *H. M. Crawford and B. Gottlieb, Baylor University, College of Dentistry, Dallas, Tex.* We observe cases in which the development of hypercalcified, opaque barriers in the dentin associated with caries, is perfect, others in which they are poor. Clinically these differences are of utmost importance. It was found that in the presence of signs of strong acid action, the barrier of hypercalcification was absent, or present only on the 2 lateral sides and absent in the direction of the pulp. The following explanation is offered. In the presence of sufficient acid, the calcium salts are kept in solution and can not be precipitated as an opaque barrier. The acidogenic microorganisms proceed quickly along the dentinal tubuli and the acid produced prevents precipitation. Along the cross connections, laterally, they proceed with difficulty. Less acid is produced here, and the precipitation of an opaque, hypercalcified wall is possible. Likewise, if less acid is produced in the direction of the pulp, a barrier develops here. A small amount of acid becomes self limiting by producing a barrier to the further invasion of microorganisms.

VI. FOURTH SESSION, MORNING, MARCH 17; ABSTRACTS 26–37

26. THE CHARACTER AND DISTRIBUTION OF CARIOUS LESIONS IN THE MOLAR TEETH OF SYRIAN HAMSTERS. *Paul H. Keyes, University of Rochester, Rochester, N. Y.* Information on the character and distribution of carious lesions in the molar teeth of Syrian hamsters was obtained by placing over 125 animals on a diet of whole wheat flour 20%, corn starch 25%, confectioners sugar 20%, whole powdered milk 30%, and powdered alfalfa 5%. Rations and tap water were available ad libitum. Groups were sacrificed about every 10 days over an 81 day period. Miscroscopic evidence of caries activity was detectable in animals on the diet 10 days and macroscopic lesions were seen after twenty days. Cavities increased in size and number thereafter. Lesions were of subgingival and coronal origin. The most extensive lesions were associated with collections of plaque-like material in the gingival crevice. Evidence was found that im-

paction of fibrous material in interproximal spaces and in occlusal grooves may predispose the animals to parodontal disturbances and dental caries.

27. PLAQUE FORMATION, PERIODONTAL DISEASE, AND DENTAL CARIES IN SYRIAN HAMSTERS. *Paul H. Keyes and Robert C. Likins, University of Rochester, Rochester, N. Y.* Four groups of 18 hamsters (9 female and 9 male) were placed on basic ration of powdered milk 30 pts., powdered alfalfa 5 pts., brewers yeast 4 pts., and NaCl 1 pt. Group I received 40 pts. of basic ration and 60 pts. of whole wheat flour; Group II received 40 pts. basic ration, 40 pts. w.w. flour and 20 pts. glucose; Group III, 40 pts. basic ration, 20 pts. w. w. flour and 40 pts. glucose; Group IV, 40 pts. basic ration and 60 pts. glucose. Animals received ration and water ad libitum for 107 days. There was no apparent difference in health of animals in Groups I, II, and III. All had fine coats of fur. Group IV animals were somewhat smaller and often had fur matted with sugar. Final average weight were for Males I to IV: 100, 104, 99, and 94 grams; for Female I to IV: 128, 126, 127, 113 grams. Plaque accumulation, periodontal disease, and dental caries markedly increased as glucose content of diet increased. Average caries scores were: Group I—Female—3.2, Male—13.5; Group II—Female 14.6, Male—70.4; Group III—Female 49.9, Male 124; Group IV—Female 117, Male 114.7. Maxillary and mandibular second molars were very susceptible to caries. Males were more susceptible than females. Extensive accumulations of plaque material and periodontal disease were generally, but not always, associated with dental caries. Impaction of fibrous material (hairs and shavings) were frequently found in and around the teeth and may have predisposed the animals to periodontal disease and caries. To obtain additional information on the relationship of shavings to periodontal disturbances and caries activity a preliminary study was made on 10 male hamsters. Two groups of 5 animals, litter mate distributed, were housed in screen bottom cages and maintained for 60 days on a diet of 50 parts dry whole powdered milk and 50 parts glucose. Group I received 5 grams of the dry ration per day; Group II, 5 grams of the dry ration mixed with 7 cc. of distilled water. Tap water was available ad libitum. Accumulations of plaque material, found in all animals were associated with gingival recession and bone resorption. Eleven small cavities were found in animals on dry diet; 1 suspicious area was found in animals on liquid ration.

28. FURTHER STUDIES ON THE HISTOPATHOLOGY OF CARIES OF THE ENAMEL IN THE HUMAN TOOTH. *Harry E. Frisbie, University of California, College of Dentistry, San Francisco, Cal.* A further study has been made of the early development of the carious lesion in the human tooth. The specimens studied showed no apparent breakdown, the tooth surface appearing hard, but at this stage some brown discoloration is present. In order to prevent desiccation and distortion of detail, the specimen must be maintained wet even while the surface lesion is photographed. The recovery of the organic matrix demonstrates a surface involvement which widens as it involves the deeper part of the matrix. Microscopic examination shows much of the structural detail has been lost in the lesion, although islands of uninvolved enamel remain scattered. Only occasional colonies of organisms are found in the central areas, while the periphery

of the lesion shows a progressive extension of the carious process as indicated by the presence of spheroidal organisms and the tinctorial reactions of the matrix beyond the area of central lysis. A more rapid extension of the process is observed to spearhead out from the periphery of the lesion along tracts, as described in earlier reports.

29. VARIATIONS IN COUNTS OF LACTOBACILLI MADE ON SINGLE SPECIMENS OF SALIVA. *Dorothy Permar, Paul C. Kitchin, Hamilton B. G. Robinson, Ohio State University, College of Dentistry, Columbus, Ohio.* Various sources of error have lead to recognized inaccuracies in counts of lactobacilli in saliva. The range of these variations has been demonstrated by making numerous series of 8 to 27 plates from single saliva specimens, using a diluent for the saliva before plating. None of the various methods of shaking and beating the original specimen produced consistent results. Using 1 cc. of saliva in a measured amount of diluent (Jay's beef broth), 39 series of plates from 39 specimens were made. Of these, 26% gave 100% of the plates within the range of $\pm \frac{1}{2}$ the mean for that series; and 28% gave 50 % or less of the plates within $\pm \frac{1}{2}$ the mean. Since using a larger portion of the original specimen would help to reduce error due to clumping of the bacteria in the saliva, another method was tried in which the original specimen was thoroughly shaken, and 5 cc. of it were removed to 5 cc. of fluid agar. This mixture was beaten on a high speed stirrer, and from it, 1 cc. was removed to 4 cc. of Jay's beef broth, mixed and plated. A series of plates was made from this first saliva and agar mixture, using a different broth dilution for each plate. In 16 series of plates made from 16 specimens, 69% gave 100% of the plates within the range of $\pm \frac{1}{2}$ the mean, and only 6% (1 series) gave 50% or less of the plates within the range of $\pm \frac{1}{2}$ the mean for that series. While the second method improved the consistency of the counts, it, too, in one series, gave unsatisfactory results. General trends were apparent in the lactobacillus counts of individuals studied. However, these data indicated that no conclusions on hourly or daily fluctuations are justified when they are based on studies of single counts or on such series as we have made from single specimens. The variations in a single series of counts may be as great or greater than an assumed hourly or daily fluctuation.

30. THE EFFECT OF CADMIUM ON DENTAL CARIES IN THE RAT. *Henry M. Leicester, Ph.D., College of Physicians and Surgeons, Dental School, San Francisco, Cal.* Since cadmium, like fluorine, causes bleaching of the rat incisor, its possible effects on caries were studied. Rats were placed on the coarse corn caries-producing diet for 100 days and given drinking water containing 0.002% or 0.004% $CdCl_2$. The average caries index for these 2 groups did not differ greatly from that of the controls, but the range of variations was much greater in the cadmium fed rats. The number of cavities was not increased, but in many cases the rate of progress of the lesion was more rapid when cadmium was given. In a few cases, no caries was observed. This suggests that cadmium under some conditions may increase the rate of acid production. The evidence of Ginn and Volker (*Proc. Soc. Exper. Biol. Med.* 57, 189, 1944) that after 150 days rats on 0.005% $CdCl_2$ showed a great increase in caries index also favors this idea. It

is possible that the teeth contain enough cadmium to stimulate phosphatase activity, a property of cadmium in minute amounts. Feeding cadmium during the period of calcification of rat teeth did not affect the subsequent development of caries in any unusual way.

31. NERVE FIBER DEGENERATION FOLLOWING TOOTH REMOVAL. *B. O. A. Thomas, D.D.S., M.S., Ph.D, Columbia University, New York, N. Y.* After removal of 1 or more mandibular teeth in the cat, transverse and longitudinal sections of the inferior alveolar nerve (in situ in the mandible) were studied, after staining with osmic acid or silver nitrate. Post-operative survival periods ranged from 4 to 21 days. Their results of nerve fiber counts from cross-sections indicate: (1) there is considerable variation in the number of nerve fibers in the inferior alveolar nerves on the 2 sides of the same animal, and in the nerves of different animals; (2) there is no appreciable or consistent decrease in the number of nerve fibers following the removal of teeth. The latter finding may be explained from a study of longitudinal sections of the inferior alveolar nerve. These demonstrate that the degeneration of the nerve after tooth removal is limited to the area from which the tooth was extracted. The fact that fibers are constantly joining the main nerve trunk in these regions makes it difficult to determine the point at which individual fibers are traumatized, and consequently to measure the length of the degenerated segment.

32. GROWTH OF THE HEAD FROM BIRTH TO THE THIRD MONTH OF LIFE. *Allan G. Brodie, D.D.S., Ph.D., and Manual Ortiz, D.D.S., M.S., College of Dentistry, University of Illinois, Chicago, Ill.* This report covers findings made on 135 consecutive births at the Research and Educational Hospital, University of Illinois. The study was conducted by means of cephalametric roentgenograms and exposures were made every second day, beginning with the first day (about 20 minutes after birth) during the period of lying-in, and at 2 weeks intervals thereafter. Five Caesarean deliveries were included as controls. Examination of the X-rays revealed that the molding of the head by the birth canal resulted in a marked diminution of its antero-posterior dimension with an increase in vertical height. This is made possible by bending upward of the occipital squama from the posterior margin of the foramen magnum and by a backward bending of the divided squama of the frontal bone. Recovery from birth molding occurred in 3 days on the average although difficult births and instrument deliveries took somewhat longer. The heads of the normal births and the Caesarean deliveries attained the same mean dimensions by the third month.

33. PROGRESS OF YELLOW PIGMENTATION AND ACID ACTION IN ENAMEL AND DENTIN. *R. Boelsche and B. Gottlieb, Baylor University, College of Dentistry, Dallas, Tex.* We know that in enamel caries is spearheaded by either acid action or yellow pigmentation. If the invasion of the enamel develops along the prism sheaths, the micro-organisms which produce acid form the spearhead and the yellow pigmentation follows. If, however, an lamella which can be invaded is present, the yellow pigmentation forms the spearhead and acid action follows. In dentin acid action always spearheads, never yellow pigmentation. When a hypercalcified barrier stops the progress of acid producers the yellow pigmentation producers may catch up.

34. THE FORMATION OF SECONDARY DENTIN. *B. Gottlieb, Baylor University, College of Dentistry, Dallas, Tex.* It is generally believed that stimuli are carried from the exposed fibrils to the pulp. These stimuli result in formation of secondary dentin which separates the center of irritation from the pulp. The first layer of secondary dentin has, in most cases, no tubuli and no fibers. If the usual description of the mechanism were true, the first thin layer of secondary dentin would prevent any stimulus from reaching the pulp. Formation of secondary dentin would stop but we know that it goes on. The odontoblasts under areas secondary dentin are always far poorer than in the region of normal, slow dentin formation. Having nothing to do with the formation of dentin matrix they are separating the dentin forming connective tissue from the dentin itself thus slowing down the dentin formation. Any damage to the fibers results in means damage to the corresponding odontoblasts of which they are parts. Damaging the separating layer gives the connective tissue a better chance to form more dentin. Thus we have to understand that the formation of secondary dentin is not caused by stimulation of the forming tissue but by damaging and partly eliminating the separating layer of odontoblastsi n that way dentin dedosition is made more efficient. (Published in *J. D. Res.* **25**: 29, 1946)

35. THE MORPHOLOGY OF THE PULP CANALS OF THE PRIMARY MOLARS (A PROGRESS REPORT ON THE MANDIBULAR SECOND MOLAR). *Ralph L. Ireland, D.D.S., M.S., University of Nebraska, College of Dentistry, Lincoln, Neb.* The form of the pulp canal in the cervical portion of the mesial and distal roots resemble sa dumb-bell. In this area the pulp canal resembles the surface form of the root. The buccal and lingual ends of the canal are small (0.3 mm.–0.8 mm.), almost equal in size and connected by a narrow concave bar (0.1 mm.–0.3 mm.). In some specimens the connecting bar is present in only the cervical area (1 mm. long), while in others it tapers down to the middle or apical area. The round buccal and lingual ends continue down the roots parallel to each other, becoming smaller as they approach the root apex. It was not uncommon to find these canals opening on the inner side or surface of the root.

36. ATOMIC VERSUS MOLECULAR OXYGEN. *Balint Orban, Chicago College of Dental Surgery, Chicago, Ill.* In comparing the action of atomic and molecular oxygen upon the bismuth sulfide precipitates in the gingiva (bismuth line) we have an in vivo test for the action of two different forms of oxygen. The statu nascendi oxygen (atomic oxygen) oxidizes the insoluble black bismuth sulfide into soluble white bismuth sulfate and thus the bismuth line disappears. Molecular oxygen, insufflated into the gingiva from medicinal tank oxygen, fails to oxidize and does not remove the bismuth sulfide precipitate.

VII. FIFTH SESSION, AFTERNOON, MARCH 17; ABSTRACTS 37–45

37. THE PERIODONTIUM, PANCREAS AND BLOOD SUGAR LEVELS IN EXPERIMENTAL DIABETES. *Irving Glickman, Tufts College Dental School, Boston, Mass.* A histological study was conducted on 121 young adult male and female rats to determine whether a relationship existed between the condition of the periodontal structures, pancreas changes and blood sugar levels in experimental diabetes. Diabetes was induced by subcutaneous injection with a 5% aqueous solution of

alloxan in a dosage of 200 mg/kg body weight. Blood sugar levels were determined using the Folin-Malmos method approximately one week after injection with alloxan and also at time of sacrifice when possible. In 33 animals with chronic diabetes (blood sugar levels 200-740 mg/kg) pancreas changes were limited to the beta cells of the islets of Langerhans and varied in severity from slight vacuolar degeneration of the cytoplasm, to necrosis with loss of cellular outline, degranulation of the cytoplasm and nuclear pycnosis. In numerous animals of this group the pancreas presented no microscopically detectable changes. In addition, a correlation was not always observed between the severity of hyperglycemia and pancreas pathosis. Comparison of microscopic findings in the periodontal tissues with hyperglycemia and pancreas pathosis in individual animals with chronic diabetes revealed the following: a) Differences in severity of hyperglycemia and/or pancreas pathosis were not reflected in the incidence or severity of gingival changes. b) Varying degrees of nonspecific osteoporosis of the alveolar bone were exhibited in 39% of the animals. Neither the incidence nor severity of the destructive bone changes was specifically correlated with the presence or degree of hyperglycemia or pancreas pathosis.

38. THE CAPACITY OF PERIODONTAL VESSELS ON PRESSURE AND TENSION SIDES OF TEETH MOVED BY ORTHODONTIC APPLIANCES. *John Revesz and S. W. Chase, Western Reserve University, Cleveland, Ohio.* Using the method of weighed paper cutouts, numerical values were obtained for comparison of the volume of the periodontal membrane and the capacity of its vessels on the pressure and tension sides of orthodontically moved teeth in the macaque. For the canine teeth moved by the fairly strong force of a lingual expansion appliance the periodontal membrane on the pressure side was compressed to less than half (45.8%) the volume of that on the tension side. The vessels of the pressure side, however, had a capacity of only about one-tenth (10.5%) of those on the tension side. Similar analyses were made for molars moved by the force of separating wires and for teeth not treated orthodontically serving as controls.

39. CHANGES IN THE SALIVARY VOLUME AND pH WITH CHANGES IN OXYGEN CONTENT OF THE INSPIRED AIR. *S. Wah Leung, D.D.S., School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Partial anoxia was produced in human subjects by decreasing the oxygen tension in the inspired air. Breathing of the gas mixture, held in a 500 liter spirometer, was made possible through a system of tubing and a mask covering the nose and mouth. Saliva was collected through the mask by means of a special funnel arrangement. Measurements of the salivary rate of flow and salivary pH showed definite changes in both of these properties with a trend toward lowering magnitude. These changes appear to be subject to individual variations.

40. CHEMICAL STUDIES IN PERIODONTAL DISEASE. III. THE RATE OF PUTREFACTION OF SALIVARY PROTEINS UNDER VARYING CONDITIONS. *M. Berg, Ph.D., Dan Y. Burrill, D.D.S., L.S. Fosdick, Ph.D., School of Dentistry, Northwestern University, Chicago, Ill.* Saliva collected from individuals with normal periodontal tissues and from individuals with pathological periodontal tissues was allowed to putrefy under aerobic and under anaerobic conditions, and with and

without added casein. After incubation for 1 hour, 3 hours and 24 hours, samples were removed and analyzed to determine the amount of hydrolysis of proteins as indicated by the formol titration, and to determine the amounts of indole, tryptophane, tyrosine and sulphides formed. The oxidation reduction potentials (Ec) and the odor concentration were determined and the EH and RH values were calculated. The results of these determinations were analyzed statistically and it was found that the differences in degree of hydrolysis, indole, sulphides and concentration of odor were significant. Incubation for 1 and 3 hours under anaerobic conditions with added casein proved to give the most significant differences. A factor involving all of the significant variables was derived. (To be published in *J. D. Res.*)

41. CHEMICAL STUDIES IN PERIODONTAL DISEASE. IV. THE RATE OF PUTREFACTION OF SALIVARY PROTEINS AS AN INDEX OF PERIODONTAL DISEASE. *M. Berg, Ph.D., Dan Y. Burrill, D.D.S., L. S. Fosdick, Ph.D., School of Dentistry, Northwestern University, Chicago, Ill.* Saliva was collected from 100 individuals with normal periodontal tissues and from 100 individuals showing periodontal disease, and was incubated under anaerobic conditions after the addition of casein. After incubation for 1 hour and 3 hours, samples were removed and analyzed to determine the amount of protein hydrolysis by formol titration, indole, and hydrogen sulphide. The concentration of odor was measured. It was found that there was a significant correspondence between the values obtained and the condition of the periodontal tissues. It was not determined whether the higher rates of putrefaction were the cause or the effect of pathological conditions.

42. THE DISINTEGRATION OF SILICATE CEMENT DENTAL RESTORATIONS. I. THE EFFECT OF ACIDS AND SALTS ON SILICATE CEMENTS. *Paul E. Weston, B.S., D.D.S., Ph.D., University of Tennessee, Memphis, Tenn.* Even though manufacturers have done extensive developmental research and conscientious dentists have followed faithfully minute details of manipulative technic dental silicate cements are generally considered to produce temporary restorations because average life expectancy of such fillings is only about 4½ years. Failure is due mainly to solubility, or disintegration in fluids of mouth. Experiments show that the disintegration of silicate cements is due to the presence in the mouth of some patients of one or more chemical substances which will dissolve a silicate cement rapidly regardless of care and skill exercised in constructing it. Rate of disintegration was studied by suspending samples of set silicate cements in buffered solutions of hydrochloric, lactic and citric acids. In all cases rate of disintegration depends on hydrogen ion concentration, pH being a linear function of the logarithm of the time, but nature of the acid itself being of profound importance. In case of hydrochloric acid-phthalic acid mixtures, rate of disintegration found by extrapolating the line to pH 7 is of same order as that noted by other investigators for solubility in pure water. Hydroxy-carboxylic acids such as lactic, citric, tartaric, and gluconic acid dissolve silicate cements rapidly, even in alkaline solution, apparently by formation of soluble compound by chemical reaction between cement and the hydroxy-carboxylic acid. Aluminum is probably element in cement primarily responsible for reaction. Car-

boxyl group alone or hydroxyl group alone does not cause reaction. Amino acids behave similarly, but more slowly. Both original glassy cement powder and binding material formed when the cement sets are soluble, but binding material dissolves more readily. It is well known that lactic acid is produced in mouth along with other acids from the fermentation of carbohydrates and so these experiments indicate that in those persons' mouths in which such transformations take place silicate cement restorations will disintegrate rapidly even though oral fluids are neutral or alkaline, and in spite of skillful manipulation used in their construction.

43. EXPOSED DENTAL PULP AS A POSSIBLE PORTAL OF ENTRY FOR THE POLIO VIRUS. *Sidney B. Finn, A.B., D.M.D., M.S., Robert Korns, M.D., D.P.H., and A. Bahlke, M.D., M.P.H., Department of Health, Albany, N.Y.* An examination of the pulp exposure rate among 1945 polio cases and controls in Upstate New York, reveals no significant difference. Of the 70 cases of polio studied having siblings 32 or 45.7% had pulp exposures while of the 119 siblings in the families of these cases 54 or 45.4% had exposures. Of the total 151 cases of polio studied 56 or 37.1% had pulp exposures while among 148 controls consisting of all household contacts 59 or 39.8% had pulp exposure. Of the bulbar cases studied 9 or 33.3% had exposures while of the 31 siblings 12 or 38.6% had exposures. A comparison of the bulbar with the non-bulbar cases of polio reveals that of the 27 bulbar cases in the study 9 or 33.3% had exposures while of the 124 non-bulbar cases 47 or 37.9% had pulp exposures. From the data gathered in this study no evidence appears that the exposed dental pulp is a significant pathway for the entrance of the polio virus in human beings.

44. SOME OBSERVATIONS ON THE FERMENTATION OF CARBOHYDRATES BY SALIVA. *J. F. Volker and Dorcas Pinkerton, Dental School, Tufts College, Boston, Mass.* Studies have been made of the hydrogen iron concentration and titratable acidity of the various carbohydrate saliva mixtures. It has been established that the rate of fermentation of sucrose and glucose is comparable and somewhat greater than that for starch. However, the latter in turn is considerably greater than certain of the dextrins. The fermentation of the concentrations of sucrose and glucose varying from 100 mg to 10,000 mg %, incubated with saliva, has been investigated. It has been shown that through most of this range, acid production is independent of the concentration. It is believed that it is not necessary to have polysaccharides and disaccharides broken down to glucose prior to fermentation. It is conceivable that the more complex carbohydrates undergo a direct phosphorilization. The ability of wheat and navy bean extracts to inhibit the saccharification of starch has been demonstrated.

45. pH STUDIES ON ORAL MICRO-ORGANISMS. *Robert M. Stephan and Elizabeth Hemmens, The Zoller Memorial Dental Clinic, University of Chicago, Chicago, Ill.* Masses of micro-organisms growing on teeth produce a rapid drop in pH when carbohydrates are ingested, but similarly rapid pH changes do not occur with the low microbic cell concentrations usually employed in acid production tests with culture media or saliva. In order to compare the ability of different micro-organisms to produce pH changes such as occur on teeth, pure cultures of micro-organisms isolated from plaques were grown, and concentrated by

centrifugation in a buffer resembling saliva to approximately the concentration of microbic cells on teeth. A glass electrode was used to determine pH, and experiments were run at 37.5°C. of 14 types of micro-organisms tested, almost all produced a rapid pH drop when carbohydrate was added, but only some gave a rapid pH rise when pyruvate, lactate, or urea was added as a substrate. Differences in pH curves occurred not only with different micro-organisms but also with different cell concentrations of the same micro-organisms, and with different substrate concentrations as well as with different substrates. The complex nature of these pH curves suggests that many more enzyme systems are involved in the caries process than merely those which produce acid.

VIII. Sixth Session, Evening, March 17, Abstracts 46–49

46. Remarks of retiring president. *Wallace D. Armstrong, Ph.D., University of Minnesota, Minneapolis, Minn.*

47. Inaugural address. *Samuel W. Chase, Ph.D., Western Reserve University, Cleveland, Ohio.* (See p. 151.)

48. Experiences and observations in war zones. I. Italy. *Isaac Schour, D.D.S., Ph.D., University of Illinois, Chicago, Ill.*

49. Experiences and observations in war zones. II. Europe. *H. Trendley Dean, D.D.S., United States Public Health Service, Bethesda, Md.*

IX. Abstracts 50–86, Papers Read By Title

50. Lack of influence of an estrogenic hormone on teeth. *W. D. Armstrong, Ph.D., M.D.*, University of Minnesota, Minneapolis, Minn. Eleven mature rats were given 5 micrograms estradiol dipropionate in sesame oil by subcutaneous injection on alternate days for 53 days. Fifteen similar control animals were treated in the same manner and on the same schedule with sesame oil. Results as means and standard errors of means: Percentage ash of humeri; 67.89 ± 0.123 (control), 70.56 ± 0.076 (experimental); Percentage ash of incisor teeth, 78.94 ± 0.131 (control), 79.60 ± 0.123 (experimental). It is concluded that the estrogenic hormonal influence on teeth is not as marked as on the skeleton.

51. Pathological calcification in the gingivae. *William F. Barnfield, D.D.S., M.S., University of Indiana, School of Dentistry, Indianapolis, Ind.* Calcified bodies in the gingivae originate by metabolic activity. Material for this study was selected from 120 sections. The development of fully calcified and ossified bodies is traced by showing successive stages. Spheroids originate in old and hyalinized granulation tissue. Calcium is present at an early stage. The spheroids clump to form larger calcified bodies. The local and general conditions, which bring about calcification, are discussed.

52. Induced alveolar bone destruction and endocrinal disturbances in the rat. *William F. Barnfield, D.D.S., M.S., University of Indiana, School of Dentistry, Indianapolis, Ind.* It was found that fastening of wire around the cervix of the 4 first molar teeth was a controlled, uniform method of irritating the alveolar bone. The wire was fastened by twisting the ends at the mesial aspect of the tooth. After 7 weeks, the wires were found to be in much the

same condition as when they were placed. Results thus far indicate that this is a controlled method of injury, similar to calculus in the human, and that it will be useful in the study of experimental periodontitis in rats with alloxan diabetes.

53. CHANGES IN THE INCISORS OF HYPOPHYSECTOMIZED FEMALE RATS AFTER DIFFERENT POSTOPERATIVE PERIODS. *H. Becks, M.D., D.D.S., D. A. Collins, D.D.S., M.S., M. Simpson, Ph.D., M.D., H. Evans, M.D., College of Dentistry, Institute of Experimental Biology and George William Hooper Foundation for Medical Research, University of California, San Francisco and Berkeley, Cal.* 140 female hypophysectomized rats were sacrificed at intervals between 6 and 664 days after operation. Earliest changes in upper incisor as seen in roentgenogram consisted of thickening of labial and lingual dentin at expense of pulp chamber. These alterations occurred at 14 and 87 days respectively after hypophysectomy and were only constant and pathognomonic symptoms observed. Distortion of apical labial curvature, distinct folding of upper incisor alone, and folding of both upper and lower incisors were first seen 100, 144, and 205 days respectively after hypophysectomy. Neither of these symptoms were pathognomonic. Folding of upper incisors alone was most frequently seen. It occurred in 46% of hypophysectomized rats 144 or more days after operation. Folding of lower incisors was present in only 13 of the 40 rats which showed foldings of upper incisors. It never occurred alone; i.e., without simultaneous folding in upper jaw. Various degrees of folding were found to be expression of delayed eruption. Histologic observations were limited to animals with long postoperative intervals. Most outstanding change in cellular structures occurred in ameloblastic layer. Regressive changes were not uniform for all animals but were directly proportionate to severity of distortion or folding of apical tissues. Degeneration of ameloblasts in localized areas led to enamel hypoplasia and in most severe form to enamel agenesia leaving connective tissue in direct contact with dentin surface. Degeneration of the odontoblasts began in the incisal portion of incisor and reached the apical end only in animals which showed advanced folding. Marked diminution in vascularity of the pulp tissue also characterized advanced changes. Increase in thickness of cementum was observed histologically in incisors of all hypophysectomized rats and did not vary with the postoperative interval (316 to 644 days after operation).

54. RESPONSE OF THE TIBIA OF HYPOPHYSECTOMIZED RATS TO GROWTH HORMONE AND THYROXIN AFTER LONG POSTOPERATIVE INTERVALS. *H. Becks, M.D., D.D.S., M. Simpson, Ph.D., M.D., H. Evans, M.D., and C. W. Asling, M.D., College of Dentistry, Institute of Experimental Biology, George William Hooper Foundation for Medical Research, University of California, San Francisco and Berkeley, Cal.* I. Female rats hypophysectomized at 26 to 28 days of age were maintained for 275 to 550 days after operation. Three groups of 5 rats each then received daily injections of anterior pituitary growth hormone for 5, 15, and 30 days, respectively; a fourth group served as uninjected controls. Histologic examination of proximal epiphysis of control tibias showed cessation of chondrogenesis and osteogenesis; the epiphyseal cartilage was sealed by bone from diaphyseal marrow cavity. Following growth hormone injections for 5 days epiphysis showed activation of chondrogenesis, moderately active osteo-

genesis, and beginning breaking up of the "sealing-off bone". After 15 days of injections, chondrogenesis and osteogenesis were both more active, and remaining "sealing-off bone" had been moved down into diaphyseal marrow cavity. After 30 days, chondrogenesis and osteogenesis showed the high degree of activity observed in young and growing normal rats. II. Under similar operative and postoperative conditions, 4 groups of 10 rats each received respectively daily injections of pituitary growth hormone, thyroxin, a combination of the 2, and no treatment (controls). After 39 days of injections the animals were sacrificed. Length of tibia was measured at autopsy. Mean for control group—30.2 mm. After thyroxin injections average—1.2 mm. longer; after growth hormone injections average increase—2.9 mm. over control, and after injection of the 2 in combination—5.7 mm. Mean values in all injected groups were significantly greater than controls. Average width of proximal epiphyseal cartilage in histologic sections was 215 u. for controls. No appreciable widening followed administration of thyroxin. After growth hormone injections cartilage width was 60% greater, and following injections of growth hormone + thyroxin, 75% greater than that of controls. Following administration of thyroxin mild activation of chondrogenesis and osteogenesis occurred, accompanied by a breaking up of "sealing-off bone." After growth hormone injections, very active chondrogenesis, capillary erosion, and osteogenesis were seen (Part I). The combination of growth hormone and thyroxin injections was followed by a degree of activation slightly exceeding that which resulted from growth hormone alone. Growth hormone and thyroxin are synergistic in reactivating chondrogenesis and osteogenesis in the cartilage disc of the proximal tibial epiphysis in hypophysectomized rats following long postoperative intervals.

55. GROWTH AND TRANSFORMATION OF THE MANDIBULAR JOINT IN THE RAT. III. THE EFFECT OF GROWTH HORMONE AND THYROXIN INJECTIONS IN HYPOPHYSECTOMIZED RATS. *H. Becks, M.D., D.D.S., D. A. Collins, D.D.S., M. S., M. Simpson, Ph.D., M.D., H. Evans, M.D., College of Dentistry, Institute of Experimental Biology and George William Hooper Foundation for Medical Research, University of California, San Francisco and Berkeley, Cal.* 41 female rats were hypophysectomized between 23 and 30 days of age. After postoperative intervals ranging from 0 to 617 days they were injected with either growth hormone, thyroxin, or both. Injection of growth hormone into hypophysectomized rats after long postoperative intervals produced dramatic resumption of growth of all components of mandibular articulation. This renewed growth was characterized by reactivation of chondrogenesis and osteogenesis, and compared favorably with growth observed in 20 day old normal rat. In rats which received injections of thyroxin only response observed was slight increase in matrix of condylar cartilage. Combination of growth hormone and thyroxin injections was followed by stimulation of both chondrogenesis and osteogenesis; however, not all of features which characterized mandibular joint of young growing animal were restored. Vacuolization of the cartilage cells and new trabeculae formation in neck of condyle lagged behind that produced in rats injected with growth hormone alone.

56. PRELIMINARY REPORTS ON THE USE OF SODIUM FLUORIDE IN A PROPHY-

LACTIC CLEANING MIXTURE AND IN A MOUTHWASH. *B. G. Bibby, H. Zander, Mary McKelleget, A.B., D.H., Bertha Labunsky, D.H., Tufts College Dental School, Boston, Mass.* A study in which 3 dental prophylaxes using a 1% sodium fluoride-pumice-hydrogen peroxide mixture were given to the teeth on one side of the mouth of 47 children showed an overall reduction in new caries of approximately 43%. A similar study in which 2 fluoride prophylaxes were given to 95 children reduced caries by approximately 25%. It is not believed that the difference in these results is fundamentally the result of the number of treatments given. In both instances caries reduction in the upper teeth was much greater than in the lower teeth. The use, thrice weekly, of an acid (pH4) mouthwash containing 0.1% sodium fluoride for 1 year did not reduce caries activity in 31 dental students below that of 15 using a control fluoride free mouthwash or 39 using no mouthwash. It is suggested that the negative results obtained with the mouthwash may be the result of the patients being too old to be benefited by fluoride therapy. (To be published in *J. D. Res.*)

57. FURTHER OBSERVATIONS ON THE VITAL STAINING OF DENTIN AND ENAMEL. *Charles F. Bodecker, D.D.S., and William Lefkowitz, D.D.S., Columbia University, School of Dental and Oral Surgery, New York, N.Y.* To date, 224 teeth have been stained vitally in the following animals: dogs, sheep, calves, cats, monkeys and in man. Conclusions were based on observations made on the diffusion of water soluble eosin, congo red, Sudan IV, borax carmine, India ink, methylene blue, potassium permanganate, argyrol and silver nitrate. The 2 latter were found best for vital staining purposes. A number of techniques were used in vital staining; sealing dry dyes: (1) in experimentally prepared cavities in sound dentin; (2) on the dental pulp; and (3) on tbe surface of the enamel. Observations of this material refutes the controversial issues in the report of Zander and Smith (*J. D. Res.* **24:** 121, 1945) who used only silver nitrate in solution. The fact that no solid dyes were used may have led to erroneous conclusions. The accumulated data support the following conclusions: 1. Dyes penetrate metamorphosed dentin (resulting from aging or the peripheral irritation) to a variable degree, but much less profusely than through young dentin. 2. Dyes reaching the pulp from a cavity drilled in dentin, are redistributed to surrounding dentin and enamel; this indicates that dental lymph originates from the pulp ("secondary vital staining"). 3. Dye particles such as argyrol and potassium permanganate are carried far from the dye deposit, thus presenting further evidence of efferent and afferent currents of dental lymph in teeth of dogs and young human teeth. 4. The presence of the dental lymph diffusing in 2 directions may explain the mechanism of dentin metabolism. (To be published in *J. D. Res.*)

58. "THE ILLUSORY SHEATH OF NEUMANN IN DENTIN". *Charles F. Bodecker, D.D.S., Columbia University, School of Dental and Oral Surgery, New York, N. Y.* Two new techniques have thrown light on this long sustained controversial problem. The most common conception of Neumann's sheath is that dentinal tubules are lined by highly acid resistant, modified dentin matrix, which can be isolated by macerating dentin with 25 to 50% nitric acid. Others claim that

these isolated structures are not linings of tubules but represent dentinal fibers. In order to test the latter conception, dentin sections were incinerated up to 1100 degrees C. for 11 min. and then decalcified under coverglass by Kitchin method. The decalcification is automatically halted before complete destruction of dentin, due to saturation of acid with mineral salts. Tubular structures were isolated by this heat-decalcification technique, similar to those procured by maceration alone. This suggested that the isolated structures are not dentinal fibers as these are regarded as being protoplasmic in nature. Thus, apparently the existence of Neumann's sheath was further substantiated. This conclusion was again refuted by the use of another method, that of over-decalcifying dentin, embedding it in paraffin and preparing exceedingly thin sections (5). These showed the partial destruction of the matrix, surrounding the dentinal fibers, the supposed site of Neumann's sheath. The structures that persisted were dentinal fibers. Conclusions. The structures surviving the dentin maceration are tubular; these do not represent Neumann's sheaths, but are tubular processes of the odontoblasts. The highly resistant character of the dentinal fibers necessitates research in dentin physiology. (To be published in *J. D. Res.*)

59. GROWTH AND TRANSFORMATION OF THE MANDIBULAR JOINT IN THE RAT. I. NORMAL FEMALE RATS. *D. A. Collins, D.D.S., M.S., H. Becks, M.D., D.D.S., M. Simpson, Ph.D., M.D., H. Evans, M.D., College of Dentistry, Institute of Experimental Biology and George William Hooper Foundation for Medical Research, University of California, San Franisco and Berkeley, Cal.* The mandibular joints of 69 normal female rats of the Long-Evans strain, ranging from 5 to 465 days of age at autopsy were roentgenographed, and studied histologically. At 5 days of age, the condyle and neck of the ramus were composed of hyaline cartilage. After this time the cartilage model was gradually replaced by bone until only a thin covering of cartilage was left which, with a fibrous border, formed the articular surface. After approximately 258 days the neck of the condyloid process consisted of an almost solid mass of bone; the cartilage matrix was highly calcified except for a narrow embryonic zone located at the articular end of the condyloid process.

60. GROWTH AND TRANSFORMATION OF THE MANDIBULAR JOINT IN THE RAT. II. HYPOPHYSECTOMIZED FEMALE RATS. *D. A. Collins, D.D.S., M.S., H. Becks, M.D., D.D.S., M. Simpson, Ph.D., M.D., H. Evans, M.D., College of Dentistry, Institute of Experimental Biology and George William Hooper Foundation for Medical Research, University of California, San Francisco and Berkeley, Cal.* The mandibular joints of 79 hypophysectomized female rats were studied histologically. The animals were between 23 and 30 days of age at the time of operation and were sacrificed at postoperative intervals ranging from 4 to 645 days. The changes in the mandibular joints of these hypophysectomized rats were similar to but occurred very much earlier than those in normal rats. Chondrogenesis was already considerably slowed down 4 days after the operation. The advanced stages of calcification characteristic for the 258 day old normal rat had been reached already at 28 days after hypophysectomy. Even at 645 days after the operation, a narrow embryonic zone of cartilage persisted. The persistence of this primordial tissue suggests a capacity for renewed growth.

61. PENICILLIN IN THE TREATMENT OF INFECTIONS OF THE FACE AND JAWS. *Thomas J. Cook, D.D.S., work done at 20th General Hospital, India.* Twenty-eight cases were treated with penicillin and 23 controls of essentially similar lesions comprised the clinical material. All except 2 of the patients were hospitalized and thus represented lesions of more than average severity. Due to low stocks of penicillin at the time of study, only 2 cases were treated prophylactically, but use in this manner is strongly advocated. Cases treated; Chronic Osteomyelitis—10 cases, Acute Osteomyelitis—7 cases, Soft tissue infections—11 cases. (Ludwig's angina, Cellulitis of dental origin, Pericoronal abscess and Lymphadenitis). Summary: 1. Penicillin is a valuable aid in the management of infection of the face and jaws, but is not a substitute for sound surgical measures. 2. Acute and chronic osteomyelitis without sequestration is quickly healed; that type with sequestrum improves only after their removal. 3. In odontogenic infections and their sequelae, penicillin administration produces rapid subsidence and allows early extraction with little risk of post-extraction cellulitis and lymphadenitis. 4. It is a powerful prophylactic agent and should be widely used for this purpose.

62. OBSERVATIONS ON THE TREATMENT OF ORAL INFECTION WITH SULFATHIAZOLE GUM. *L. L. Eisenbrandt, University of Kansas City, Kansas City, Mo.* Preliminary studies indicate that sulfathiazole gum may be used to treat Vincent's infection. In our hands a limited number of cases improved markedly in 24 hours as observed by clinical and laboratory methods. The method of prescribing was 8 tablets (0.25 gm. each) per day for 3 days, then 3 tablets per day for 3 days. Each tablet was chewed 1 hour. Some cases received no other treatment; others, scaling of the teeth. There was no evidence of toxicity. One chronic case failed to clear up, although there was improvement.

63. THE PURPOSE OF THE ODONTOBLASTS. *B. Gottlieb, Baylor University, College of Dentistry, Dallas, Tex.* Since it was shown definitely that the odontoblasts furnish the fibers, but do not form the dentin, the function of the odontoblasts appears questionable. The tubuli which house the fibers are readily able to carry infection to the pulp. The function of the odontoblasts seem to be keeping open a draining system through which fluid may reach the dentino-enamel junction. The enamel is fixed to the dentin by the tufts, which apparently need moistneing in order to retain their functional ability. Clear separation of enamel from dentin is normally impossible but if teeth are allowed to dry, the enamel often peels off in grinding. Such a smooth peeling off of enamel from dentin may sometimes be observed in cavity preparation of pulpless teeth. It can also be seen in hereditary opalescent dentin. In both these conditions, the flow of tissue fluid to the dentino-enamel junction is eliminated. (Published in *J. D. Res.* **25**: 25, 1946)

64. HEREDITARY OPALESCENT DENTIN. *B. Gottlieb, Baylor University, College of Dentistry, Dallas, Tex.* Hereditary opalescent dentin is characterized by obliteration of the pulp canal and enamel chipping off. Examination of ground sections revealed that the dentinal tubuli are regularly arranged only in the

first formed mantel dentin. The later formed dentin, which obliterates the pulp canal has but a few irregularly arranged tubuli. This indicates that the odontoblasts have disappeared in all teeth at an early time and may suggest systemic factors. We now explain the formation of surplus secondary dentin as resulting from damage to the odontoblasts through their fibers. Normally, the connective tissue of the pulp, which forms the dentin matrix, is slowed down in its activity by the separating layer of the odontoblasts. If they are damaged their retarding power is removed and more dentin is deposited. If all the odontoblasts degenerate because of some systemic cause the whole pulp canal is obliterated by unhampered dentin formation. No suggestion can be offered concerning the systemic cause. The elimination of flow of tissue fluid to the tufts, which are supposed to fix the enamel to the dentin impairs their condition and permits the peeling off of the enamel from the dentin.

65. DENTAL CHANGES OF RATS AND HAMSTERS IN VITAMIN E DEFICIENCY. *Humberto Grandos, Karl E. Mason and Henrik Dam, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* When reared from weaning to 5 to 6 months of age upon an E-deficient diet, containing 18% lard and 2% cod liver oil, Florida cotton rats showed no loss of incisor enamel pigment, whereas hamsters exhibited complete depigmentation of maxillary incisors such as seen after about 60 days in the albino rat. There exist marked differences in the ability of E-deficient diets high in fat to inhibit enamel pigment deposition in these rodents. Histologically, the jaws of albino rats reared for 5 to 333 days of age upon this diet, or one in which cod liver oil replaced the 18% lard, exhibited a progressive atrophy of the enamel organ and an increasing deposition of acid-fast pigment in the bone marrow fat, in macrophages at the perivascular and fibrous zones surrounding the enamel organ, and, later, in those that had migrated to the atrophied enamel organ itself. Essentially the same histological changes occurred in rats when 20% cod liver oil was replaced by its equivalent of highly unsaturated fatty acids of the oil. Similar feeding of the medium and low unsaturated fatty acid fractions, or the non-saponifiable fraction, of cod liver oil produced no such changes; neither did an E-deficient diet devoid of fat. Vitamin E therapy progressively restored enamel organ structure and enamel pigment deposition but, except for changes secondary to macrophage migration, did not appreciably influence the pigment deposition in the adjacent periodontal tissues. This latter deposition of acid-fast pigment seems identical to that observed in the adipose tissue of the same animals, as recorded elsewhere. Both the processes of incisor depigmentation and deposition of acid-fast pigment in soft tissues require the presence of highly unsaturated fatty acids as well as inadequacy of vitamin E in the diet, but may depend upon different cellular mechanisms for their production.

66. A METHOD OF ELIMINATING GINGIVAL PIGMENTATION. *Isador Hirschfeld, D.D.S., and Leonard Hirschfeld, A.B., D.D.S., Columbia University, New York, N. Y.* This is a preliminary report describing a painless method by which unsightly brown or black pigmentation may be quickly removed from the marginal and alveolar gingivae. The pigmented area is chemically cauterized once and a

pink, normal healed surface results within a few days. Several cases of Negro and Caucasian patients are presented with progressive photographs; no pigmentation has returned so far—2 cases have been observed for more than a year.

67. PRELIMINARY REPORT ON THE PHYSIOLOGY OF ARGINASE-ARGININE METABOLISM. *W. G. Irons, D.D.S., A.B., M.S., College of Dentistry, University of California, San Francisco, Cal.* A series of procedures were developed for obtaining an extract of bovine, pig, and dog livers containing arginase. The product obtained was equivalent in potency to 1.82 of the Hunter-Downs unit. A series of white mice (Hamilton strain) were used in the experiment for evidence of reaction. The observations established a possible connection of the arginase-arginine rôle with the processes of repair, regeneration, and tumor growth. Further experiments are in progress for, 1, purification of the enzyme; 2, histological and physiological action of both enzyme and substrate on normal tissue; 3, their influence on normal and abnormal reparative processes; 4, the variations in the normal occurrence of these substances in the tissues of various types of animals.

68. COMPARATIVE ACTIVITY OF ARGININE, GUANIDINE AND THYROXIN ON GROWTH OF TISSUE CULTURES. *W. G. Irons, D.D.S., A.B., M.S., College of Dentistry, University of California, San Francisco, Cal.* Arginine, guanidine, and thyroxin were separately incorporated in graduated amounts of the media of tissue cultures. The cultures were observed to learn which tolerated the concentrations of the substances introduced. The tissues were selected from growing cells of ectodermal, mesodermal, and endodermal origin. From the reactions observed it was evident that all 3 substances exerted a stimulative effect. The sperm of guinea pigs, having an arginine content, when introduced stimulated the growth of connective tissue cultures. Thyroxin seemed to influence the growth in size of the cells, while arginine and guanidine seemed more effective on the nuclear contents. Using determined amounts of arginine and guanidine, sperm activity was maintained for an average period of 17 days.

69. BLOOD STUDIES IN PERIODONTOCLASIA, II. *Maxwell Karshan, Ph.D., Benjamin Tenenbaum, B. S., D.D.S., Frances R. Karlan, B. S., and Harold J. Leonard, B.S., D.D.S., Medical and Dental Schools, Columbia University, New York, N. Y.* The possibility that systemic factors may play a part in the etiology of periodontoclasia has been investigated further. Blood and urine were analyzed in 67 patients with incipient to advanced periodontoclasia, and in 32 controls who were free from this condition. Serum was analyzed for total calcium, inorganic phosphate, phosphatase, total protein, albumin (globulin is difference between total protein and albumin), total cholesterol, free cholesterol, sodium, chloride, vitamin A, carotene, cholesterol-cephalin, flocculation; oxalated plasma was analyzed for Vitamin C; whole blood was analyzed for sugar, non-protein nitrogen, urea nitrogen, uric acid, erythrocyte sedimentation rate, hemoglobin, and erythrocyte, leukocyte and differential counts. Basal metabolism was also determined. Statistically significant differences between means of control and periodontoclasia groups were found only for phosphatase, total protein, globulin, sedimentation rate, leukocyte count and basal metabolism.

On basis of urinary analysis, there was no evidence of subclinical deficiency of thiamin, riboflavin or nicotinic acid in either group.

70. SOME OBSERVATIONS ON THE USE OF STREPTOTHRYCIN IN PROTECTING CHICKEN EMBRYOS AGAINST INFECTION BY SALIVARY BACTERIA. *Arlette Lawyer and Basil G. Bibby, Tufts College Dental School, Boston, Mass.* Study was designed to test whether chicken embryos might be useful as a means of evaluating usefulness of antibacterial agents intended for use in close proximity to dental pulp. Eggs were incubated at 37°, opened on the 11th day and injected on 13th. Chorio-allantoic membrane was inoculated with 0.5 cc. of saliva and 0.5 cc. of streptothrycin (250 units per cc.) injected intravenously. All 5 of the embryos not protected with streptothrycin died. Eighteen of 22 injected with fresh streptothrycin survived. The percentage of survival diminished progressively in 63 embryos treated with solutions stored for 3, 6, 8, 10 and 13 days at room temperature. Histological examination of sections from the surviving embryo revealed no abnormal tissue reactions. Comparisons were made of effect of local applications of streptothrycin and Morson's creosote on 80 chorio-allantoic membranes infected with 0.5 cc. of saliva. Full strength, 6000, 600, 250, and 120 unit concentrations of streptothrycin, and full strength, 50%, 25%, and 1% creosote were used. Streptothrycin and Morson's creosote in high concentrations were injurious to embryo tissues. However, only streptothrycin exerted pronounced bactericidal effect in dilutions well tolerated by tissue. Comparisons were made of the effects of streptothrycin and penicillin injected into allantoid space of embryos. Neither 0.5 cc. of 250 unit solutions of penicillin or streptothrycin produced changes in 9 embryos. However, 0.5 cc. of 10,000 unit concentrations in 10 embryos caused death of 5 of 10 of the streptothrycin treated embryos and 3 of 10 penicillin treated embryos. These showed comparable pathosis, indicating that comparable concentrations of streptothrycin and penicillin have similar effects on chick embryos.

71. SOME OBSERVATIONS RELATING TO THE USE OF STREPTOTHRYCIN IN THE MOUTH. *Arlette Lawyer, Basil G. Bibby and Helmut A. Zander, Tufts College Dental School, Boston, Mass.* To test in vitro effectiveness of streptothrycin on mouth organisms, agar plates and half-plates containing progressive dilutions of streptothrycin were streaked with salivary organisms. 25 units per cc. of streptothrycin in horse blood agar prevented the growth of all organisms. Critical dilution just permitting growth of streptococcal types was approximately 0.4 until per cc. Yeast grew at dilution of 3.1 per cc. After 15 days storage units at room temperature, 250 unit solutions lost their effectiveness. Weak bacterial growth occurred on anaerobically incubated plates containing 25 units per cc. Ice box stored solutions were effective for 19 days. Freshly extracted carious teeth immersed in 250 unit streptothrycin solutions for 2 hour periods gave negative cultures from carious dentin and tooth surfaces. To compare usefulness of streptothrycin and Morson's creosote as cavity dressing zinc oxide mixtures containing reagents were imbedded in agar plates. Both inhibited growth of salivary organisms through zones of same width. Similar zinc oxide mixtures containing 250 units of streptothrycin and creosote were each sealed in 3 freshly

prepared sound cavities and 2 carious cavities and incubated for 48 hours. All sound cavities were sterile, carious cavities dressed with creosote yielded mixed cultures, but 2 treated with streptothrycin were sterile, even throughout layers of carious dentin 2 mm. thick. To determine whether use of streptothrycin in deep cavities would have toxic effect upon pulp, cavities prepared in 3 anterior teeth of young dog were filled with zinc oxide containing 250 units of streptothrycin per cc. Corresponding teeth were filled with creosote and zinc oxide and with zinc oxide. After 12 days, the teeth were removed from living dog and prepared for histological study. Pulps remained perfectly healthy in areas corresponding to streptothrycin treated cavities, but there were signs of mild irritation in pulps of teeth treated with creosote. As an alternative approach to testing the effect of bactericidal concentrations of streptothrycin on the pulp, experiments, reported elsewhere, were carried out using chicken embryos. Preliminary clinical tests showed reduced growth from 2 carious cavities treated with zinc oxide streptothrycin (250 unit for 3 and 5 days), sterile cultures from a similar cavity dressed with streptothrycin for 1 and 4 days. A root treatment with zinc oxide streptothrycin showed no periapical reaction and an alveolotomy dressed with streptothrycin in agar responded favorably although sterility was not maintained.

72. EXPERIMENTAL AMELECTOMY. *William Lefkowitz, Harry Shapiro, and Charles F. Bodecker, Columbia University, Faculty of Dentistry, New York, N. Y.* Previous studies on the effects of surgical removal of the dental papilla in vivo of partially formed teeth demonstrated that calcification of the enamel was arrested. In order to further investigate source and mechanism of enamel calification, the enamel organ was surgically removed from the incisal half of partially formed teeth of dogs. Specimens were taken at various subsequent periods. This report deals only with the ground section study. Examination of control specimens at the age of experimentation shows that in the matrix state, the pattern of calcification follows that of formation. Similar specimens stained with Von Kossa suggest that the dentinal tubules are the purveyors of calcifying elements. Amelectomized teeth, permitted to erupt, showed incomplete formation of the incisal half of the enamel. Clinical, histologic and Grenz-ray examination demonstrates that calcification of the experimental area is complete. All experimental teeth were malposed erupting lingual to the arches.

73. PREVENTION OF DENTAL CARIES BY BRUSHING THE TEETH WITH NATURAL AND SYNTHETIC FLUORAPATITE. *J. F. McClendon (with clinical assistance of Aris Carpousis), Hahnemann Medical College, Philadelphia, Pa.* A comparison was made of previously reported Tennessee brown rock phosphate (split-silk phosphate from H. D. Ruhm, Mt. Pleasant, Tenn.) containing 3.77% of iron and 7% silica and a synthetic fluorapatite free from iron and silica. Thirty students were given the synthetic fluorapatite and developed 0.57 new cavities per man per year, 40 students were given the Tennessee fluorapatite and developed 0.5 new cavities per man per year and 80 students used as controls developed 1.5 new cavities per man per year. The possible explanation is that fluorine from the surface of the apatite crystal exchanges with the hydroxyl groups of the hydroxyapatite on the cleaned tooth surface.

74. THE NON TOXICITY FOR ADULTS OF FLUORINE IN TOOTH POWDER. *J. F. McClendon and Wm. C. Foster, Hahnemann Medical College, Philadelphia, Pa.* The question has been raised as to the danger of fluorine-containing tooth powders. One of the authors has swallowed 1 gram per day of tooth powder containing 4% fluorine for 1 year and the other for 3 years. On examination of our bones by x-rays no osseous changes could be detected in the hand or spine by Drs. N. F. Ludwick and J. S. Lehman, radiologist. We each excreted 10 milligrams of fluorine per day in the urine during this period.

75. PREVENTION OF DENTAL CARIES IN RATS BY BRUSHING THEIR TEETH. *J. F. McClendon and Wm. Foster, Hahnemann Medical College, Philadelphia, Pa.* We have brushed the teeth of rats every day for 4 years with gritty or fine powder containing fluorine. Dental caries in rats has been attributed to the mechanical injury produced by hard bodies forced into the sulci between the cusps of the teeth. On the contrary, the gritty powders were more effective than fine powders in preventing dental caries in rats.

76. THE DESTRUCTIVE EFFECT OF CITRATE VS. LACTATE IONS ON RATS' MOLAR TOOTH SURFACES, IN VIVO. *F. J. McClure, Ph.D. and Stanley J. Ruzicka, D.D.S., Dental Research Section, Division of Physiology, National Institute of Health, U. S. Public Health Service, Bethesda, Maryland.* Enamel and dentin of rats' molar teeth were exposed to the action of dilute solutions of sodium lactate and sodium citrate, in vivo, pH of drinking fluids being respectively in 3 separate studies as follows: pH 5.5–5.6, pH 6.5–6.7 and pH 7.0–7.2. The destructive effect of lactate and citrate drinking fluids was studied by comparing weights of teeth of test rats vs. controls, as well as by scoring the tooth surfaces on the basis of an arbitary grading system. In all studies, the weight and scoring data, accompanied by ground sections, gave conclusive evidence of an extremely destructive effect of the citrate ion over the lactate ion under practically neutral conditions, pH 5.5–7.2. The action of citrate is explained as due to the binding of the calcium of the dentin and enamel by citrate to form a soluble calcium citrate complex—a process which is essentially a non-acid decalcification. Although the destruction of dental tissues by citrate bears no resemblance to caries, the results suggest that in considering the etiology of dental caries less emphasis be placed on acid decalcification and possibilities of a non-acid decalcification, brought about by bacterial metabolites, be given more attention in dental caries research. The results also suggest further studies in vivo regarding surface effects of other anions in addition to citrate, as well as effects of certain foods and beverages, on tooth surfaces. (Published in *J. D. Res.* **25:** 1, 1946)

77. EFFECTS OF VARIATION IN THE MERCURY-ALLOY RATIO UPON THE AMALGAM FILLING. *Ralph W. Phillips, B.S., Indiana University School of Dentistry, Indianapolis, Ind.* Among the many variables involved in the manipulation of amalgam is the proper proportioning of the mercury and alloy. The purpose of this investigation was to determine the importance of strict adherence to the recommended ratio. This was done by determining the actual per cent of residual mercury and by testing the physical properties. Six alloys of varying grain size were used in this study. Three different mercury-alloy ratios were employed—(1) 15% less mercury than recommended (2) recommended ratio (3)

15% more than recommended. By chemical analysis it was found that as the ratio of mercury in the original mix is increased, the per cent of residual mercury also increases proportionally—an average increase of 1 to 1.5% for each additional 15% increase in original mercury ratio. The 1 hour crushing strength decreased an average of 20% when the mercury ratio was increased above that recommended by manufacturer, and there was an average loss of 7% for the 24 hour strength. The flow increased, in some cases 7%, as mercury ratio was increased. These findings were same for both hand and pneumatic condensation and for each individual alloy. The results on physical properties tests substantiate the chemical analysis showing that as more mercury is used in the original mix, more mercury will remain in the filling.

78. STUDIES ON DENSITY OF CASTINGS AS RELATED TO THE POSITION IN THE RING. *Ralph W. Phillips, B.S., Indiana University, School of Dentistry, Indianapolis, Ind.* The purpose of this investigation was to determine whether the position of the pattern in the ring (the distance from the bottom of the pattern to the end of the ring) is an important factor to be considered in obtaining complete and dense castings. It has been suggested by some that the pattern should be placed close to the end of the ring in order to facilitate escape of air from the mold during casting. Results on over 600 castings indicate: 1. When air pressure machine is used, if the minimum pressure is at least 10 pounds, there was no detectable difference in castings which were placed close to the end of the ring ($\frac{1}{4}''$) as compared to those placed so that a greater portion of investment separates the mold and the end of the ring ($\frac{1}{2}''$). 2. With the centrifugal machine, double the number of turns is required to secure dense castings when the pattern is farther from the end of the ring. If this distance is great enough, it is impossible to secure complete and dense castings regardless of the centrifugal force used. The position of the pattern is, therefore, of definite importance when a centrifugal machine is used. 3. With the pattern place $\frac{1}{2}$ inch from the end of the ring, the actural flow of air through the mold, as measured by gas flow meter, is only $\frac{1}{4}$ as rapid as when this distance is reduced to $\frac{1}{4}$ inch. 4. Microscopical sections show this porosity to be entirely apart from shrinkage porosity and is quite extensive in areas where this back pressure is being exerted.

79. EFFECTS OF SODIUM HEXAMETAPHOSPHATE ON TEETH. *Ralph W. Phillips and Maynard K. Hine, Indiana University School of Dentistry, Indianapolis, Indiana.* Kerr and Field (*J. D. Res.* **23:** 313, 1943) reported recently that use of saturated solution of sodium hexametaphosphate ($Na_2(Na_4PO_3)_6$) as a mouth rinse will prevent deposition of calculus and stain upon teeth for appreciable periods of time. They also reported that this solution caused rapid dissolution of silicate fillings, but no visible changes in tooth structure even after immersion for 6 months. Because control of calculus deposits is an important problem in periodontia, it seemed worthwhile to check carefully the possible harmful effects of sodium hexametaphosphate on teeth. Specimens studied included 8 samples of enamel and 7 of dentin cut from freshly extracted non-carious teeth, 14 crowns of intact teeth, and 12 intact teeth mounted to expose only crowns and about 1 mm. of cervical cementum. Constant weights of these samples

were determined by a technic developed by Phillips and then each specimen was suspended in a solution of sodium hexametaphosphate for short periods of time. (In this technic a change of 0.2% is probably experimental error.) Immersion of 8 small samples of enamel in 20% sodium hexametaphosphate for 20 days showed an average weight loss of 10.3%. Other changes in weight were as follows: 7 samples of dentin in 20% solution for 20 days lost an average of 39.4%, 6 crowns of teeth in 20% solution for 20 days lost an average of 2.3%, 4 teeth in 5% solution for 20 days lost an average of .35%, 4 teeth in 5% solution for 40 days lost an average of .475%, 4 teeth in 1% solution for 20 days lost an average of .212%, 8 crowns in 1% solution for 20 days gained an average of .152%. This study indicates that concentrated solutions of sodium hexametaphosphate can cause a measurable weight loss of dentin samples and slight loss of enamel samples. In weak solutions, however, the weight loss of teeth was negligible after 40 days of immersion. This suggests that the use of concentrated solutions of sodium hexametaphosphate is questionable, but weak solutions are probably relatively safe to use. The effectiveness of weak solutions in oral calculus prevention is now being tested clinically.

80. ORTHO-PHENYL-PHENOL IN ROOT CANAL THERAPY. *P. F. Rosenstein, D.D.S., Houston, Texas.* Infected root canals of 134 teeth were treated with a 10% solution of Ortho-phenyl-phenol in coconut oil soap, which reduced the surface tension of the material. After mechanical cleaning of canals, the agent was applied on paper points saturated with the solution and in all cases a negative culture was obtained after treatments. Ortho-phenyl-phenol is not a deodorant, but in a number of cases its properties as a local anesthetic were demonstrated. New growth of bony tissue was observed at the apex of the teeth where the roentgenograms had shown bony destruction before treatment. Superiority of this drug over others in common use is indicated by the results of treatments to date.

81. COMPARATIVE ELECTRON MICROSCOPIC STUDIES OF TOOTH SURFACES. *Robert Scott and S. Wah Leung, D.D.S., School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* A replica technique which permits the electron microscopic study of teeth without subjecting them to the high mechanical pressure, temperature or vacuum of other methods is described. By this process the teeth may be preserved for future study. The additional use of metal shadow casting demonstrates a means of increasing contrast in the final image. These methods were applied to a study of different portions of the external surface of the crowns of teeth. Photographs illustrating the results obtained are included.

82. BILATERAL DENS IN DENTE. *W. F. Swanson, D.D.S., University of Pittsburgh, School of Dentistry, Pittsburgh, Pennsylvania.* This account of bilateral dens in dente is, so far as the author knows, the first to be reported in dental literature. All previously reported cases were unilateral.

83. EFFECT OF ACUTE PANTOTHENIC ACID DEFICIENCY ON THE TIBIA OF YOUNG RATS. *W. W. Wainwright, D.D.S., M.S., College of Dentistry, George William Hooper Foundation for Medical Research, M. M. Nelson, Ph.D., Institute of Experimental Biology, University of California, San Francisco and Berkeley, Cal.*

Male and female rats were placed on pantothenic acid deficient diets (*Am. J. Ortho. and Oral Surg.* **31:** 406, 1945) at day of birth. Marked retardation of growth and poor condition of the animals resulted from the deficiency. Osseous changes were studied in the tibia of young rats sacrificed beginning at 21 days of age. Chondrogenesis, osteogenesis, and hematopoiesis were markedly decreased in the region of the proximal epiphysis. The width of epiphyseal cartilage in the deficient group was approximately half that of the purified diet control animals which were exceeded in turn by the normal control animals by about one-third. "Sealing off" of the epiphyseal cartilage by bone occurred as early as 70 days of age in the deficient animals.

84. Eosinophilic granuloma. *Ellen K. Wellensiek, B.S., D.D.S., Houston, Tex.* A case is reported of a 4 year old child with eosinophilic granuloma of Hand-Schüller-Christian's disease. In the early stages of the disease the case was diagnosed malignant neoplasm. It was not until a year later that the lesions in the oral cavity were thought to be correlated with the general manifestations. With this in mind further x-ray and histological examinations were made. These new findings entirely eliminated the original diagnosis of malignant neoplasm, and proved conclusively that the patient had the rare disease of eosinophilic granuloma.

85. The efficacy of cavity varnishes in preventing pulp irritation from silicate cements. *H. A. Zander and Irene Vissotzky, Tufts College Dental School, Boston, Mass.* Three cavity varnishes were tested by in vivo studies in 100 dog teeth and 37 human teeth. Cavities were prepared on the labial surface of these teeth to approximately the same size and depth. All cavities were filled with silicate cement except that in 62 dog teeth and 21 human teeth one of the varnishes was applied before insertion of the filling. The dogs were sacrificed or the teeth were extracted at intervals varying from 1 to 15 weeks after cavity preparation and filling. The teeth were then prepared for histological study. The teeth which had cavity varnishes applied before they were filled with silicate cement show the same type of inflammatory pulp reaction as comparable teeth which had silicate fillings without varnish application. However, the severity of the reaction is not as marked underneath the varnished cavities as it is underneath the ones without varnish.

86. Sculpture in orthodontics. *B. L. Gainsforth, D.D.S., M.S., State University of Iowa, College of Dentistry, Iowa City, Iowa.* Many orthodontists have written of the importance in dentistry of facial art and esthetic concept of the face. If, as Wuerpel inferred, beauty is a condition that cannot be described by any anatomic or physical law there is a problem in passing to the students concepts of the acceptable face needed to correct the unacceptable. This problem was pursued from the sculptural standpoint, using sculpture as "third dimensional drawing". Fifteen life size heads were made, 10 being cast in plaster as permanent records. These were interpreted as masses and plains. The dimensional change of a face during orthodontic treatment and growth were compared with expressional changes. If a purely scientific study is to be made of dimensional change, it is important that exact positioning of the head be made during

the taking of records before and after treatment. If the records are photographic, even the lighting should be standarized because a very slight shifting of a photographic floodlight will bring out new forms, subordinate others and show an entirely different looking individual. We are engaged in the business of impressionism, the changing and proportioning of structures in a manner which will impress others favorably or unfavorably. It seems worth while, then, to make a study of the impressionistic effect which certain dimensional changes will bring about, that we may help our patients to enter the adult world with a minimum of esthetic and functional handicaps.

X. Executive proceedings

A. Saturday Morning Session, March 16, 1946

The Twenty-fourth General Meeting of the Association was called to order by President Armstrong at 9:30 A.M., March 16th, 1946 in the Fifth Floor Reception Room of the Hotel Continental, Kansas City, Mo., and declared in session for the transaction of routine business and the reception of reports of committees and officers. After a brief greeting and welcome from President Armstrong the minutes of the preceding meeting were offered in the form of a reprint of the Proceedings of the 1945 (Twenty-third) Curtailed, War-time General Meeting.

On motion of the Secretary-treasurer, in the absence of corrections or additions, the minutes as printed were unanimously approved.

The Ballot for the 1946-1947 Active Officers was then declared open and tellers appointed to receive and count the votes.

Between the Twenty-third and Twenty-fourth General Meetings, the Council selected its nominees for the official ballot and legally notified the membership of its choice, appointed March 16th and 17th, 1946 as the time for the Twenty-fourth General Meeting, selected the Hotel Continental, Kansas City, Mo. as the place, and directed the arrangements to be made in collaboration with American Association of Dental Schools and the Omicron Kappa Upsilon Honorary Fraternity, as has been customary. On motion these activities of the Council were ratified and approved unanimously.

The report of the Treasurer of the Committee in charge of the Wm. J. Gies Journal of Dental Research Endowment Fund was received and the committee formally thanked for its generous interest and successful efforts to promote the welfare of the Journal. As of May 1st, 1945, the capital fund was valued at $40,906.72 and as of March 1st, 1946 at $43,281.44. This represents a gain of $2,244.72 for the ten month period.

The Secretary-treasurer reported receipts of $886.25 between the date of the last general meeting and March 1st, 1946; of this, $486.50 was allocated to the Journal of Dental Research and $46.00 to the Chicago Section. Disbursements for the same period were $221.78. The William J. Gies Special Endowment Fund now has a total of $1402.98. On motion these reports were accepted and ordered printed in the Proceedings.

The Necrology Committee (George B. Denton, Chairman) reported the deaths of R. G. Hutchinson, Jr., and Paul R. Stillman (both charter members), Otto Loss, Henry A. Miller, Elkan Sanders and Charles Tuller. The complete biographies will be published at a later date. At the request of the president, the members in attendance stood with bowed heads in their honor and in appreciation of their many contributions to the good of dentistry and oral research.

President Armstrong then addressed the Association and specifically proposed the addition of a general symposium to the scientific program of each annual meeting.

B. Council Sessions

Saturday Evening, March 16th. On motion a committee of three was appointed to examine and evaluate the nominations for membership and report back to the Council, Sunday. (Committee: Dean, Hall, Hatton.) With reference to President Armstrong's symposium proposal, a committee of three was authorized to prepare and submit a symposium for the program of the next general meeting and annually thereafter till discontinued. (Proposed Committee: W. D. Armstrong, T. J. Hill, J. Frank Hall.) In line with the recommendation of Past President Dean in his 1944 address, a committee of five was authorized to examine the methods of administration and accounting in handling the funds now belonging to the Association and to report to the Council and Association such changes, if any, that should be made in these procedures. (Committee: Bunting, Hall, Hatton, Hill and Waugh.) A motion to authorize the Secretary-treasurer to prepare and distribute formal certificates of membership was lost. The petition of a Norway Group for the authorization of a Norway Section was approved in principle and passed to the full Council for final action. The Council authorized President Armstrong to indicate by telegram and letter the approval of the bills now before the Congress for the promotion and support of dental research. On motion the Council recommended the remission of the 1946-1947 dues only of those members who reside outside of North America. On motion the Association was advised to set the annual dues as has been customary recently at the rate of $5.00 per year for the 1946 calendar year and for that portion of the 1947 year up to the date of the next general meeting; of this amount $1.00 is to be assigned to the budget of the Secretary-treasurer, and $4.00 is to be appropriated for the support and publication of the Journal of Dental Research. A budget not to exceed $500.00 was unanimously approved for the operation of the office of the Secretary-treasurer for the 1946-1947 Association year, the same to be ratified at the Sunday executive sessions and the details to be submitted to the whole Council by mail.

Sunday Morning Session, March 17th. Received the report of the tellers indicating the election of all Council nominated candidates, and referring the question of honorary officers to the Council with power to act. Unanimously approved the names of those persons listed elsewhere as having fully satisfied the constitutional qualifications for memberships and worthy of submission to the

Association for a vote. On motion authorized the continuation of a Council Committee to consider and evaluate nominations for membership and directed that nominations for membership shall be in the hands of this committee not later than the first of February preceding the general meeting at which the election will be made.

CLOSING EXECUTIVE PROCEEDINGS

Approved and adopted all acts and recommendations of the Council as reported above, including approval of the remission of the dues of members not resident in North America, and of a Secretary-treasurer's budget not to exceed $500.00 for the 1946-1947 Association year. By a written ballot unamimously elected to membership the persons listed elsewhere and whose applications had received Council approval. Voted to add to the program of this meeting in the read by title sections, additional items by B. L. Gainsforth, W. G. Irons and Philip F. Rosenstein. By a unanimous vote fixed the due for 1946 and that portion of the Association 1947 year before the 1947 general meeting at the rate of $5.00 for a 12 month year, per member. By a rising vote the persons and agencies who had contributed so much to the success of this general meeting were heartily thanked and highly commended, and specifically:

Dean Rinehart and the staff of the Kansas City Western Dental School of the University of Kansas City.

The local chapter of the Omicron Kappa Upsilon Honorary Fraternity.

The Chamber of Commerce and the two competent secretaries who assisted at the Saturday morning registration.

The excellent projection service supplied essayists.

The students who supplied part of the entertainment for the Sunday Evening Session.

The Hotel Continental and staff.

OFFICERS AND MEMBERS ELECTED

ACTIVE OFFICERS: President—*Samuel W. Chase*, Western Reserve University. President-elect—*Harold C. Hodge*, University of Rochester. Vice-president—*Allen G. Brodie*, University of Illinois. *Secretary-treasurer—Edward H. Hatton*, Northwestern University. Editor—*Hamilton B. G. Robinson*, Ohio State University. Members of the Publication Committee—*Sumter S. Arnim*, Ohio State University and *J. Frank Hall*, University of Indiana. Trustee—*Leuman M. Waugh*.

MEMBERS ELECTED: Bethesda, Md.—*Stanley J. Ruzicka*, USPHS. Boston, Mass.—*Harold Berk* and *Abraham E. Nizel*, Tufts Dental School. Chicago, Ill.—*Thomas B. Coolidge*, Zoller Dental Clinic, Chicago U. Cleveland, Ohio—*John Revesz*, Western Reserve U. Houston, Texas—*Harold B. Younger*. The Hague, Holland—*Ch. F. L. Nord*, Sec. F. D. I. Nashville, Tenn.—*Clifton O. Dummett*, Meharry Med. School. Newark, N. J.—*Marcu Brucker*, N. J. Health Dept. New Orleans, La.—*Alton K. Fisher* and *Duane W. Lovett*, Loyola U. New York, N. Y.—*Seymour J. Kreshover*, New York U. Rochester, N. Y.—*Humberto Granados*, Rochester U. Stockholm, Sweden—*Gustav Dahl*. St. Louis, Mo.—*Barnet M. Levy*, Washington U. Total, 15.

Resignations since last reported: Ronald Barber, A. T. Newman, W. M. Rogers. Total membership as of March 20th, 1946, 644.

REPORT OF SECRETARY-TREASURER (FINANCIAL)

Receipts, 1945 Calendar Year

Dues, general fund portion	$93.20
Dues, Journal of Dental Research portion	486.50
Dues, Chicago Section	46.00
Interest on general fund	8.12
Den. Art and Den. Science fund	234.93
Interest of Wm. J. Gies Endowment Fund	17.50
Total	$886.25

Disbursements for Expenses, 1945 Calendar Year

Postage and mailing	$62.32
Letter and clerical service	48.25
Printing	59.25
Supplies and stationery	24.93
Travel	27.03
Total	$221.78

MEMBERS VOTING AT ANNUAL MEETING

Aisenberg, G. M. Anderson, Appleton, Arnold, Ash, Austin, Bauer, Bear, Becks, Berger, Beube, Bibby, Bier, Blayney, Blumenthal, Bodecker, Boucher, Boulger, Boyle, Bradel, Brandhorst, Brashear, Brauer, Broadbent, J. B. Brown, J. C. Brown, D. A. Boyd, Bunting, Burket, Burn, Carr, Chase, Cheyne, Clawson, Clough, Cohen, Coolidge, Cox, Coy, Cross, Dahlberg, Davis, Deakins, H. T. Dean, Marguerite Dean, R. A. Dean, Deatherage, Dickson, Diefenbach, Dietz, Dressell, Easlik, Easton, Ralph W. Edwards, F. C. Elliott, R. G. Ellis, Elvove, Ennis, I. A. Epstein, Sidney Epstein, Erikson, Fairbank, Fee, Figg, Fleming, Fox, H. E. Friesell, Gabel, Gafafer, Gilkison, Glickman, Goldberg, Goldman, Gottlieb, Granger, Grossman, Gurley, Hahn, Hall, Hansen, Harrison, Hatton, Higley, Hill, Hirschfeld, Hodge, Holliday, Hubbell, Jasper, Jay, Jordan, Jump, Kaiser, Kaletsky, Kaplan, Karshan, Keilty, Kerr, Kitchin, Klaffenbach, Kniessner, Knutson, Koch, Kotanyi, Krogh, Land, Lalonde, Lasater, Lasby, Lefkowitz, Little, P. C. Lowery, Luckhardt, Lynch, Main, Manly, Mann, Margolis, Mason, Massler, McBride, McCall, McCauley, McClendon, McClure, McCollum, McCrea, Merkeley, Merritt, Midgley, Milhon, Millberry, Miner, Mary Moore, T. E. Moore, A. H. Mueller, Emil Mueller, R. E. Myers, Neuwirth, F. B. Noyes, H. J. Noyes, Cartel, Oblatt, O. A. Oliver, Orban, O'Rourke, Oxner, Paffenbarger, C. E. Palmer, Perrott, Peyton, Phillips, Price, Prinz, Rabkin, Radusch, Rittershofer, H. B. G. Robinson, J. Ben Robinson, Rosenstein, Romnes, Ryder, Sarnat, Scherer, Schram, D. B. Scott, Shapiro, Shell, Sicher, Silberstein, Simon, Simmonds, Sinclair, Skillen, Skinner, Sloman, Sorrin, Souder, Speidel, Stafne, Steadman, Steinmeyer, Stephan, H. A. Swanson, W. F. Swanson, Sweet, N. O. Taylor, W. E. Taylor, Teuscher, Thoma, B. O. A. Thomas, H. S. Thompson, J. R. Thompson, Treloar, VanHuysen, Volker, Volland, Wach, Wainwright, Waite, Waldron, Ward, Wassermann, Waugh, Weatherford, Weinmann, Wesson, Wheeler, Willett, N. B. Williams, Willman, F. J. Wolfe, Worman, W. L. Wylie, Jr., Zander, Zegarelli, Ziskin. Total, 217.

MEMBERS IN ATTENDANCE AT ONE OR MORE SESSIONS, LISTED BY SECTIONS

Ann Arbor—Bunting, Easlik, N. O. Taylor. *Baltimore*—J. Ben Robinson. *Boston*—Bibby, Glickman, Manly, Marjerison, F. W. Morse, O'Rourke, Volker. *Chicago*—Blackwell, Blayney, Brodie, Burrill, C. W. Freeman, Hatton, Lasater, H. J. Noyes, Orban, Pomes, Romnes, Schour, Shell, Sicher, Skinner, Steinmeyer, Stephan, Teuscher, Tylman,

Wallace, Weinmann. *Cleveland*—Chase, Dressell, Hill, Wylie. *Columbus*—Arnim, Kaiser, Robinson. *Houston*—Elliott, Peyton. *Indianapolis*—Hall, Hine, Phillips, VanHuysen. *Iowa City*—Boyd, Bryan, Cheyne, Kern, Klaffenbach, R. V. Smith. *Louisville*—Myers. *Minnesota*—Armstrong, Crawford, Lovestedt, Radusch, Ray, Rudolph, Simon, Stafne, Waldron, Worman. *New Orleans*—Speidel, Peterson. *New York*—Beube, East, Hemley, Holliday, Thomas, Ziskin. *Philadelphia*—Boyle, Gabel. *Pittsburg*—Swanson, Sweeney, Wright. *Richmond*—Brashear. *Rochester*—Finn, Hodge, Keyes, McCauley. *San Francisco*—Fleming, Frisbie, Hollenbeck, Leicester, Nuckolls, Sloman. *St. Louis*—Bauer, Boling, Brady, Brandhorst, Gurley, Main, Wood. *Tennessee*—R. A. Dean, Clawson. *Washington*—Arnold, Beall, Dean, Hampp, Schoonover, Souder. *Canada, Toronto*—Mason. *China, Chengtu*—Agnew. *No Section Membership*—Blackerby, Coleman, R. W. Edwards, Eisenbrandt, Gottlieb, Gruebbel, Hooper, T. A. Keys, Levine, Molnar, T. E. Moore, Wise. Total, 115.